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JOURNAL OF NEUROSURGERY

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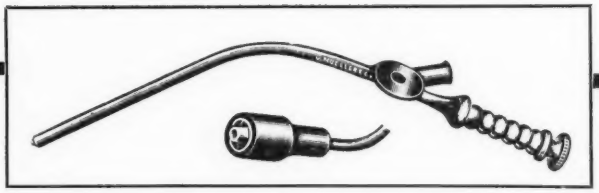

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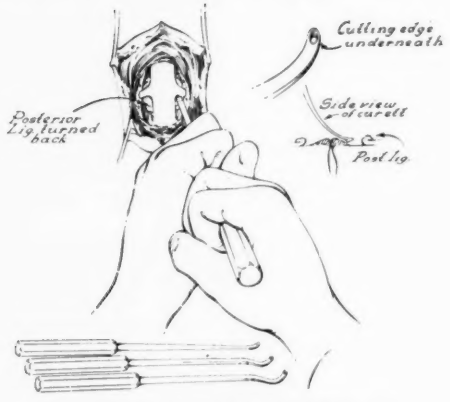

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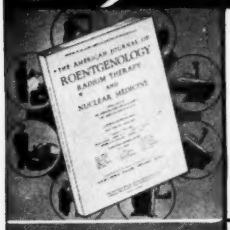

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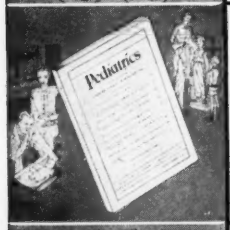

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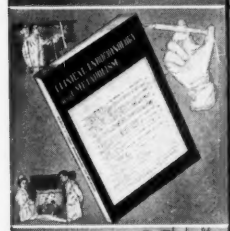

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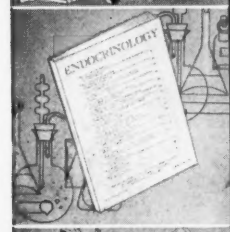

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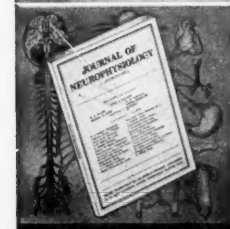

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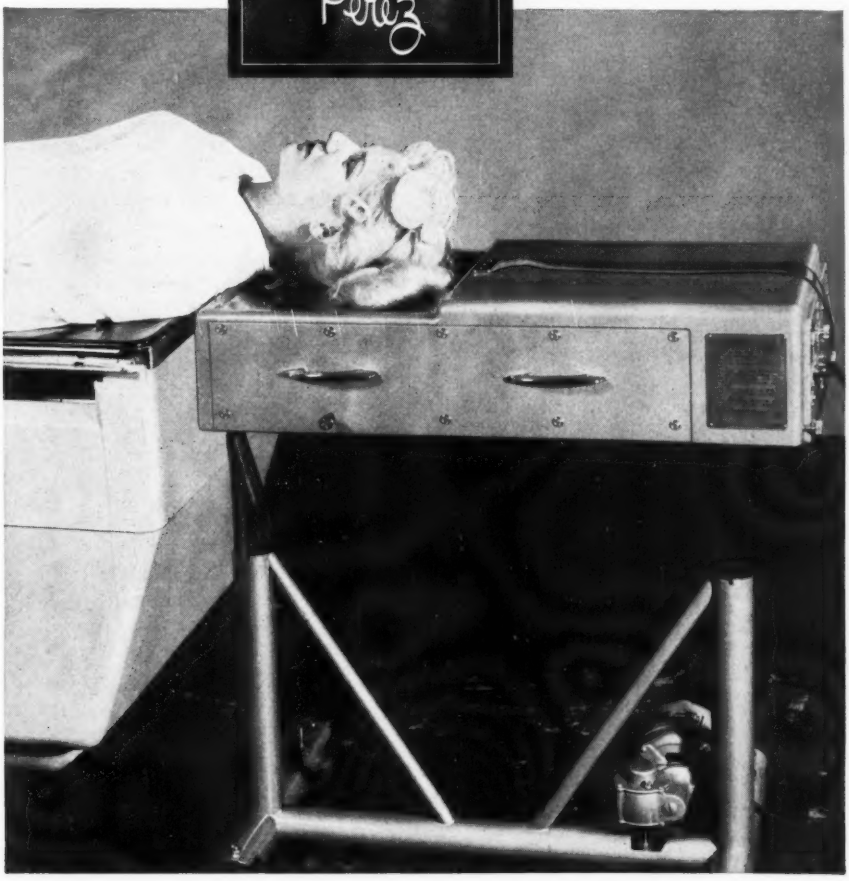

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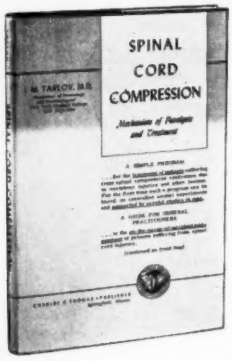

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THE PLACE OF NEUROLOGICAL SURGERY IN THE UNDERGRADUATE CURRICULUM*

LEO M. DAVIDOFF, M.D.
Albert Einstein College of Medicine, New York, New York

(Received for publication May 13, 1957)

At the time when this Society was founded, 25 years ago, its standards were already so high that they seemed to aspire to the uncompromising perfectionism that characterized the acts and deeds of Harvey Cushing himself throughout his lifetime. However, there were only 30 founding members of the Society, and in the early years the mantle of office fell more frequently on the few shoulders available to receive it. At the present time, with a membership of around 600 and the reputation of being the second oldest, and perhaps the most influential neurosurgical society in the world, it is a very great distinction indeed to be elected to be your President. I cannot fully express my gratitude to you for having so honored me.

There is no magic in the number 25, and basically no more reason to celebrate the 25th anniversary of a wedding or the foundation of a society than the 24th or the 26th. But man has an attraction for round numbers, and the division of time into millennia and centuries somehow satisfies an urge for orderliness that our psychoanalytic confrères may be able to explain. However, since none of us expects to survive a hundred years, we have accepted the common practice of dividing them into convenient fractions such as one half or one fourth. Thus we find ourselves today at a universally recognized milestone, our silver anniversary. Your Program Committee has taken cognizance of this by the beautiful cover design, and our dearly beloved Historian, Louise Eisenhardt, will undoubtedly review the achievements of the Society during its eventful existence, later on. What I should like to do, with perhaps only an occasional glance backward, is to evaluate the place of neurosurgery today as a teaching discipline to medical students.

The problem of the place of neurosurgery in the undergraduate curriculum is simply symbolic of the same problem that confronts any of the surgical specialties, or indeed all the minor and even the major disciplines. Up to 15 or 20 years ago the development of a new specialty received recognition in medical schools by the establishment of a new department, the appointment of a professor, and the allocation of a block of hours in the curriculum, which then appeared to have unlimited elasticity.

The time has been reached, however, when the expanding body of knowledge can no longer be incorporated into our teaching by the simple device of

* Presidential address, read at the Silver Anniversary Meeting of the Harvey Cushing Society, Detroit, Michigan, April 25, 1957.

stuffing more facts into the same skin. As Darley[2] put it:

"Limitations upon the human intellectual capacity preclude the addition of much more curricular material without adding to the time now currently required and limitations upon the human span of usefulness make it unreasonable to cut further into one's potential years of professional service."

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It is within this framework that I would like to develop my theme.

About a year ago, in anticipation of setting up a teaching program at a new medical school, I circularized most of the members of this Society who were heads of neurosurgical divisions or departments at other schools for their opinions concerning the place of the neurosurgeon in the medical school faculty, and the place of neurosurgery in the undergraduate medical curriculum. I also asked each of you, to whom I wrote, whether the practical working-out of the existing program at your school meets your ideas completely, or whether, if you could, you would change it to approach more closely your philosophical concept of teaching neurosurgery to undergraduates. In response to the latter, Bill Van Wagenen, with his usual caustic wit, reminded me of one definition of philosophy as: "The refined, learnèd art of ignoring fact and going wrong with confidence." Nevertheless, he and most of the others took the trouble to present me with not only the details of their own programs, but their very valuable reflections on what we are trying to accomplish and how far we are succeeding in connection with this problem.

I have received replies from the following members: Eben Alexander, Jr.; Percival Bailey; E. Harry Botterell; Jefferson Browder; Paul C. Bucy; the late Eldridge Campbell; Loyal Davis; Arthur Ecker; Edgar Fincher; William J. German; Francis C. Grant; Robert Groff; Wallace B. Hamby; Thomas I. Hoen; Sir Geoffrey Jefferson; Edgar A. Kahn; Kenneth G. McKenzie; John M. Meredith; W. Jason Mixter; Howard C. Naffziger; Eric Oldberg; Herbert Olivecrona; Carl W. Rand; Theodore Rasmussen; Bronson S. Ray; Frederick L. Reichert; Ernest Sachs; Michael Scott; R. Glen Spurling; Frank Turnbull; William P. Van Wagenen; A. Earl Walker; Arthur A. Ward, Jr.; James C. White; and Barnes Woodhall. This occasion presents me with an opportunity to thank each of you for the trouble you

took in replying. Thus, you see that the credit for what follows is yours rather than mine. My function has really been only to report and correlate your individual opinions.

It may at first sight be a far cry from grammar school to neurosurgery, but education is a continuing process, and the kind of neurosurgeons we are to become may be as dependent upon the inspirational qualities of our kindergarten teachers as upon the courses in neurosurgery in our medical schools. There can be no doubt that many a defect in professional competence may be traceable to faults in earlier education. Given the kind of material we have to work with, however, the undergraduate teacher, in the words of the late Sir Lionel Whitby:[5]

". . . should aim at producing an educated person grounded in principle and method, able to see what the whole of medicine stands for and means, trained to observe with his hands and his senses, encouraged to think logically and critically, instructed in the use of instruments of measurement, and equipped with a basic knowledge upon which he will continue to build for the rest of his professional life."

The Council on Medical Education and Hospitals of the American Medical Association, in its 56th annual report[1] warns that:

"The hope of presenting the entire subject satisfactorily in the usual course must be abandoned as an unnecessary and futile endeavor, because no individual can be expected to master all phases of medicine. Furthermore, many parts of the subject can not be taught by the faculty; they must be learned by the student through actual experience. It is an axiom that all true education is self-education. This immediately makes the student the unit of education, not the courses, the credit hours, or the faculty, and requires that the methods of instruction should be modified accordingly."

The object of teaching neurosurgery to undergraduates is, thus, not to make them neurosurgeons, but to contribute to the education of future doctors. The ideals and personality of the neurosurgical teacher are, therefore, as important as the subject matter.

This, in essence, is the gist of the opinions expressed by the majority of my correspondents.

Paul Bucy stated: "The tendency in any specialty is to place too much emphasis on it and require too much time in that particular field. I would be perfectly happy to see undergraduate instruction in neurological surgery placed on an elective basis and have teaching confined to [case work on the wards with small groups of students]. Under such a system the students who are interested would certainly attend the rounds of the good teacher, and I am not at all sure that it is worth while to bother with the ones who are not interested or with the poor teacher."

Hamby, too, is skeptical of the value of neurosurgery as an undergraduate discipline. He wrote:

"My personal concept of a neurosurgical teaching program is that its place is at the postgraduate level. The undergraduate is still at the stage of the physics major who has not yet finished calculus; exposure to atomic physics (at this stage) is

likely to be wasted, or to produce intellectual indigestion. I think we should be content to have the undergraduate know that some neurological lesions are treatable surgically."

Sir Geoffrey Jefferson stated: "I am deeply convinced that undergraduates like surgical neurology and are benefited by it. How else can they learn what can be done for the patients for which they, before long, will be responsible, and nervous affections are so very common?"

He went on to say, however: "I have assumed that students should be taught neuro-surgery, but what I really mean is the fundamentals to its uses. Taking time to give them technical details is absurd; it is a criticism against teaching that is hard to meet except by precept and example. If one assumes—and one should—that every medical student is going to be a practitioner in a small town one gets the best idea of what to teach, what the contents and substance of the teaching should be. The very best students, those that are not going into small towns but are going to be professors themselves, are not the real audience. It is nice to have them there but they teach themselves. I have said 'You can't teach anybody anything—they learn.' "

McKenzie, too, has: ". . . always felt that undergraduate teaching in neurosurgery is on the whole unsatisfactory." "I am inclined to think," he continued, "that too much time is given to it in most medical schools. . . ."

Olivecrona wrote: "Personally I do not feel it necessary that the general practitioner should have an extensive training in neurosurgery, especially as there are so many more important subjects for him. We try to let students see what neurosurgery is doing, what type of cases should be referred to a neurosurgical center, but no detailed knowledge of neurosurgery is required."

This apparently does not apply to traumatic neurosurgery in Stockholm, where a separate period for it is set aside in the course in general surgery.

Spurling declared in his letter: "My feeling has always been that nothing other than basic principles should be taught to undergraduates. For instance, we never try to teach them anything about the technical details of neurological surgery; we never give an operative clinic, nor do we talk very much about specific diseases."

Woodhall also stated: "One of our fundamental difficulties in the medical school is separating what we should teach to the undergraduate student and what we should teach to the post-graduate student; and there is little doubt that we try to do too much post-graduate teaching [in the undergraduate years]."

Thus we see that there is general agreement that definitive training in neurological surgery should be confined to the postgraduate period, and that the undergraduate should be presented only with the broad basic principles underlying the subject.

The next question, then, is how to accomplish this. Here, opinions expressed in your letters are at considerable variance. This is quite understandable, since the method of teaching varies from place to place and depends upon a number of local factors such, for example, as the quality of the medical neurological teachers and their relation to the neurosurgeons; whether

neurosurgery is a division of surgery, a joint discipline with medical neurology, or completely independent; etc.

A sampling of opinions from my correspondents makes this point quite clear:

"At Bowman Gray," wrote Eben Alexander, "the neurosurgical teaching is closely integrated with neurological teaching. Although the Section of Neurosurgery is rightly under the Department of Surgery from the administrative point of view, and although our technical work is performed in the operating room as surgeons, from the point of view of teaching and the care of patients on the wards as well as from the point of view of diagnostic methods, our closest association is with the neurologists."

He went on to say: "In some ways the teaching of neurosurgery is analogous to the teaching of Latin and Greek of old days in our colleges. There is no concrete evidence that teaching in the present day without the classics is better than, or even as good as, it was in previous days. This may be because the discipline of some of the courses such as the classics are missing."

Bailey also declared: "It seems to me that neurosurgery is so thoroughly grounded in the intellectual discipline of neurology that it is impossible to present it apart from clinical neurology, except as a technique, and such technical details are not suitable for undergraduates."

Hamby wrote: "It does not matter whether good diagnosis is taught by neurologists or by neurosurgeons, and I think we enhance the whole field by encouraging the neurologists to advance also. Of course we must go along with them and not allow ourselves to become merely their technicians, but with proper rapport between the divisions, this is not a real danger."

Jefferson, whose letter was accompanied by a reprint of an address he gave before the "First World Conference on Medical Education" in London in 1953, emphasized two points of primary importance: 1, Teaching at the bedside, and 2, the enthusiasm and dedication of the teacher. Of the one he wrote:

"The importance of bedside teaching can be tested by wondering what parts of the medical curriculum could be omitted without danger to the student's education. Almost all, most of us would reply, but this." And of the other he said: "We never try to tell them the latest thing about anything without making it clear to them that this is exciting news and making them realize the logic of why it is exciting. I have so often said that, about teaching, the great thing is to be so simple and so logical that a cat could understand it! But I do think one needs to be excited about what one is teaching. I've said, again, that I think *passion* is the most important ingredient in a teacher, that and *clarity*."

McKenzie underscored this view when he wrote, a propos of lectures in neurosurgery:

"I think the lecturer should try to put himself in the place of a family doctor possibly out in the country and then try and pass on information which would be helpful to him in placing a neurosurgical problem."

Rasmussen said: "I am convinced that the medical student's contact with a single neurosurgical patient constitutes the most efficient utilization of his time, and gives him a better insight into the way a neurological or neurosurgical problem should be approached, than he gets from several times that much exposure to a didactic lecture, or a clinical demonstration."

Michael Scott epitomized it: "Our objective for the student has been: 1. Not to confuse him with minutia. 2. To stress a systematic neurological examination as part of his medical equipment. 3. To show him how diagnostic procedures such as myelography, encephalography, ventriculography, angiography, and electroencephalography can help in diagnosis."

Turnbull, however, sounded a note of warning when he said: "I think we need to stress the neurosurgeon's viewpoint in examination which carries with it the responsibility for definitive treatment as opposed to the rather academic viewpoint that sometimes is apparent in neurological examination by a medical neurologist."

Walker, too, felt: "At the present time, with the dichotomy in teaching, there is considerable confusion in the minds of many students regarding what appear to them to be entirely different concepts between neurology and neurosurgery. Some of this confusion is caused by different emphasis by the teaching in the Departments of Medicine and Surgery, and could be cleared up by a certain amount of integration. On the other hand, there are rational and natural differences of opinion because of the difference in the approach of the neurologist and the neurosurgeon. Combined teaching, however, would clarify these differences and not leave the poor students in the middle struggling between the dogmatic and sometimes diametrically opposed statements of their instructors."

Concerning how much we would hope the students will retain from our course in neurosurgery, there is again considerable agreement.

Ward stated it as follows: "What I am basically trying to do is teach the students how to recognize disease of the nervous system, how to handle, at least in a preliminary fashion, such fairly common problems as head injury, epilepsy, headache and similar entities. They should have a pretty good concept of what the field of neurosurgery is all about and some concept of the technical difficulties that a neurosurgeon faces and something about the results that can be achieved."

White also wrote: "I would like to be able to have every man who comes through here know how to deal with an acute cranio-cerebral or spinal injury because he may well be called upon to take over after a roadway accident even if he is a dermatologist or a psychiatrist. They should also, regardless of whether they become surgeons or medicos, know the fundamentals concerning brain tumors, aneurysms, and other lesions of that sort which can masquerade in the guise of so many other medical conditions. The medical student's life, however, is too short to do this and I doubt if any school has the perfect solution."

When I first began to tally your replies, I thought that I would make a chart of the number of hours devoted to teaching our subject; in what years of the medical school it is being taught; and how much didactic as opposed to case demonstrations and bedside teaching, or "learning," as Jefferson prefers, is customary. I found the variations in this respect so great that it

soon was evident that such a tabulation would be meaningless, and it was abandoned.

However, after studying the reports of your valuable experiences and extracting from them what appeared to me to be the essence, I have worked out a tentative plan which I should like to present to you, based on your ideas plus my own preferences, and the particular conditions prevailing at our school.

First I should say that at the Albert Einstein College of Medicine the division of neurosurgery is part of the larger Department of Surgery, and quite fortuitously the head of the division of neurosurgery is also the Chairman of the Department of Surgery. We also have a very strong division of neurology, but this is in the Department of Medicine. Fortunately we are able to work harmoniously together in the clinic and the laboratories, and at the bedside. Thus the divisions of neurosurgery and neurology are able to function in a very special way as integrating forces between the Departments of Medicine and Surgery.

Our first contact with students is in the first year, during their courses in anatomy, physiology, and biochemistry, when clinical teachers in various branches are called upon to participate in a weekly exercise called "Clinical Correlation of the Basic Sciences." This continues through the second year as well, during the courses in pathology, microbiology, and pharmacology.

Beginning in the second quarter of the second year we have organized a course under the direction of the Professor of Medicine, called "Clinical Methodology." This includes instruction in history-taking, physical diagnosis, clinical pathology, and ward procedures demonstrating various techniques from bed-baths, catheterization of the urinary bladder, enemata, etc., to lumbar puncture. A wide participation by the faculty occurs in this course. Thus, for example, an internist will teach the student how to do a general physical examination, but an ophthalmologist will show him how, with specialized knowledge and special techniques, additional data may be obtained concerning the eyes. As far as the nervous system is concerned, the neurologists and the neurosurgeons share the responsibility of presenting him with its special problems. The student is not expected, as a result of this, to become a specialist in the diagnosis of diseases of the nervous system, but at least, when later in his career he hears of some esoteric procedure like sinusography of the superior longitudinal sinus he will be able to say to himself: "Where have I seen that face before?"

Beyond the second year our plans are tentative, since our first class of students has not reached the stage of clinical instruction. We expect to give no separate courses in surgery and the surgical specialties, such as neurosurgery, head and neck surgery, plastic surgery, thoracic surgery, urology, orthopedics, etc. The students in the third and fourth years will take a course in "surgery" consisting of clerkships in the In- and Out-Patient Departments of the hospital, during which they will be assigned cases which may have special problems in any of the above fields. The supervision of

the student in working-up a given case will come from a member of the surgical faculty who is a specialist in the particular field in which that patient's problems lie. Running through the third and fourth years there will be a weekly one-hour whole-class demonstration of some surgical problems including, of course, neurosurgical ones which we hope will serve as a chain to link into a total picture the experiences acquired through more personal contact with individual patients during the clerkships.

We expect to make our reading list accompanying the course quite eclectic and not necessarily exhaustive, but we do hope to stress the importance of reading original descriptions of disease whenever possible.

Let me illustrate the rationale for this kind of emphasis: In a charming little essay on "Classics of Medicine," the late Henry Sigerist[4] contrasts the description of the parathyroid glands in *Gray's Anatomy*—an excellent book by any standard—with the original description of these bodies by Ivar Sandström of Upsala.

In *Gray's Anatomy* the parathyroid glands are defined as: "small brownish-red bodies, situated as a rule between the posterior borders of the lateral lobes of the thyroid gland and its capsule. They differ from it in structure, being composed of masses of cells arranged in a more or less columnar fashion with numerous intervening capillaries. . . ."

In Sandström's classical paper of 1880 he wrote: "About three years ago I found on the thyroid gland of a dog a small organ, hardly as big as a hemp seed, which was enclosed in the same connective tissue capsule as the thyroid, but could be distinguished therefrom by a lighter color. A superficial examination revealed an organ of a totally different structure from that of the thyroid and with a very rich vascularity. . . ."

How easy it is to forget the anatomical description, and how vivid and unforgettable is the original report, I leave to each of you to judge.

This, then, is the summation of our combined thinking. Medical education has reached an impasse whereby the furiously rapid accumulation of knowledge and the limitation of time has made it impossible to include every specialized field of medical endeavor into the undergraduate teaching program, without replacing more basic material from the curriculum. Neurosurgery is among these specialties. The teaching of neurosurgery in its detailed aspects should, therefore, be consigned to teaching postdoctoral students who are planning careers as specialists in this or related fields. During the undergraduate years the neurosurgeon can and should participate in teaching, together with his neurological and surgical colleagues, by bringing before the students a knowledge of the form and function of the nervous system; something about the diseases to which it is susceptible; and a bowing acquaintance with the availability and effectiveness of neurosurgery in the case of some of these conditions. Neither the future general practitioner, nor the potential specialist, on the day when he receives the right to call himself a "Doctor of Medicine," can hope for competence in diagnosing or treating surgically even the less complicated neurosurgical ailments. He should look

upon his diploma as a special permit to enter into the great, awe-inspiring, discovered world of the science and art of medicine, with its many still uncharted regions. Let us hope that by precept and example we may be worthy to be his guide!

REFERENCES

1. Council on Medical Education and Hospitals of the American Medical Association. Medical education in the United States and Canada. *J. Amer. med. Ass.*, 1956, *161:* 1637–1678.
2. Darley, W. Horizons of medical education. *J. med. Educ.*, Jan. 1957, *32:* 15–19.
3. Pusey, N. M. Education and medicine. *Harvard med. Alumni Bull.*, Jan. 1957, *31:* 7–9.
4. Sigerist, H. E. The university at the crossroads. Addresses & essays. *New York: H. Schuman*, 1946, viii, [2] 162 pp. (see pp. 92–105).
5. Whitby, L. The challenge to medical education in the second half of the 20th century. *J. med. Educ.*, Nov. 1953, *28:* 26–33.

NEUROFIBROMAS OF THE FIFTH CRANIAL NERVE*

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JEFFERSON, in a recent review of the literature and report of his own cases, analyzed 32 instances of trigeminal neurofibromas. Baker and Bailey, Cushing and Eisenhardt, Gonzalez Revilla, Loew and Tönnis, and Davidoff and Epstein also have reported another 11 cases, so that the total number thus far reported is 43.[1–19,21–24,26–28]

PRESENT STUDY

In this communication we are reviewing 13 cases in which solitary neurofibromas (neurilemmomas) of the fifth cranial nerve were encountered at the Mayo Clinic. We have excluded all cases of von Recklinghausen's disease.

For purposes of this study, the cases were divided into three groups morphologically, according to whether the lesions arose mainly in the middle cranial fossa, subtentorially in the posterior fossa, or in both situations in more or less equal extent. However, some of the tumors that presented in either the middle or posterior fossa also had extended slightly into the other fossa.

Jefferson found that in the 32 cases he reviewed, 15 lesions were mainly or entirely tumors of the middle fossa, 11 were situated in the posterior fossa and 6 were tumors of both the middle and posterior fossas. In our series, 6 lesions were restricted primarily, if not entirely, to the middle fossa, 4 were essentially limited to the posterior fossa and 3 were both supratentorial and infratentorial.

We found that very often it is difficult to make an early diagnosis of this kind of tumor. In our series, some change in the cutaneous sensibility of the face constituted an early finding in only 6 instances, and in 2 patients symptoms and signs of involvement of the fifth nerve were absent. In only 4 cases was pain present in the distribution of the fifth nerve, and this pain was not tic-like, although it was periodic in one instance. In another case the pain extended from the region of the ear into the paratracheal and inferior mandibular region and it was also periodic.

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Based in part on a thesis submitted by Dr. Olive to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Neurosurgery. The pathologic material has been reviewed by Dr. J. W. Kernohan, Section of Pathologic Anatomy, Mayo Clinic.

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We found (Fig. 1) that the tumor occurred most frequently in the fourth decade (6 patients), next in the fifth decade (4 patients), less frequently in the third (2 patients) and in the sixth decade (1 patient).

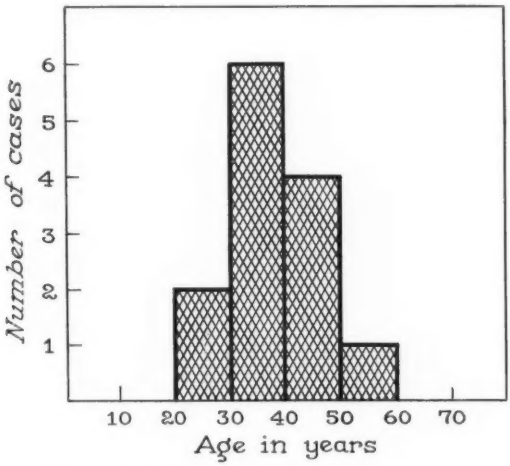

FIG. 1. Distribution of trigeminal neurofibromas according to age of patients.

SUPRATENTORIAL TUMORS

Case 1.—A white married woman 45 years old registered at the clinic in 1939, complaining of progressive left exophthalmos during the preceding 4 years. During this interval she had noticed, in the back of the neck, pain which occasionally would awaken her at night. This pain frequently would be present when she awoke, and would disappear after she was up and around.

Ophthalmologic examination revealed exophthalmos of 7 mm. on the left side. Results of neurologic examination were negative. Roentgenograms of the skull showed enlargement of the sella turcica, with erosion of the floor and the posterior clinoids. The visual fields appeared to be normal. A diagnosis of unilateral exophthalmos of undetermined origin was made. Roentgen therapy was advised and accepted. It was administered to the pituitary and retro-orbital region.

Three months later the patient returned. She said that since the last visit she had experienced repeated vomiting, pain in the left eye, blurred vision and some disturbance of memory. Ophthalmologic examination revealed slight pallor of the left optic disk and bitemporal upper quadrantic contraction in the visual fields. New roentgenograms of the skull were made; they disclosed an increase in the erosion of the posterior clinoids over the erosion that had been noticed at the previous examination. A diagnosis of a chiasmal lesion was entertained.

On April 13, 1939, left transfrontal craniotomy was carried out. An extrasellar mass was present anterior to the optic chiasm. Biopsy proved this to be a neurofibroma (neurilemmoma). Intracapsular removal was carried out and most of the capsule was removed. The tumor did not appear to invade the orbit.

The patient had a stormy postoperative course and died on the 6th postoperative

FIG. 2. *Case 1.* Tumor occupying the left side of the middle fossa. Notice relationship to the petrous apex. Specimen obtained at necropsy.

FIG. 3. *Case 1.* Compression and deformity of the brain produced by the trigeminal neurofibroma.

day. At necropsy a tumor was found to occupy the left middle fossa. The tumor was 6 by 3.5 by 3.5 cm., and on microscopic examination proved to be a neurofibroma. A nodule of the tumor compressed but did not invade the pituitary gland, and the lesion was found to enter the left retrobulbar area. The tumor arose from the trigeminal nerve in the middle cranial fossa (Figs. 2, 3 and 4).

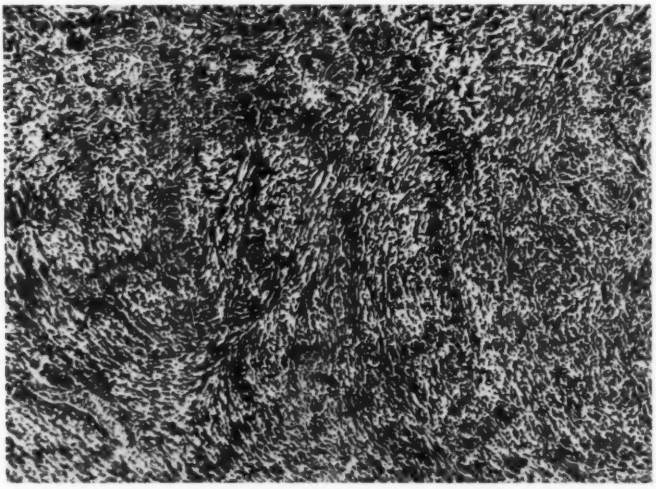

FIG. 4. *Case 1.* Left trigeminal neurofibroma (neurilemmoma). Note the typical pattern (hematoxylin and eosin, ×60).

Case 2.—A white man 21 years old registered at the clinic on Nov. 6, 1953. He said that in the latter part of 1949 he had first noticed some transient numbness in the right cheek. In the early part of 1951 the left side of the face had become numb, and he had first noticed a throbbing headache in the occiput. This headache extended forward over the left side of the face, and was aggravated by exertion, stooping and coughing. In September, 1953, he had first noticed that dizziness resulted if he changed his position rapidly. This was more marked when he was turning to the left side. In October, 1953, he had first noticed the onset of tinnitus in the region of the left ear.

Neurologic examination revealed marked hypesthesia and hypalgesia in the distribution of the left trigeminal nerve. The corneal reflex was absent; marked weakness of the masseter, pterygoid and temporal muscles on the left was noted. There was also moderate diminution of taste on the left side of the tongue. Ophthalmologic examination disclosed slight weakness of the left external rectus muscle. The ocular fundi and the visual fields were normal. Moderate hypoactivity of the left labyrinth was found. Hearing was within normal limits. Roentgenograms of the skull revealed the left anterior clinoid process to be decalcified from below and elevated, together with a sharp area of decalcification at the tip of the left petrous apex. A diagnosis of tumor of the left side of the middle fossa was made.

On Nov. 24, 1953, a left carotid angiogram was made. The left internal carotid artery was elevated and displaced medially. On Nov. 25, left transtemporal

craniectomy was carried out. The dura mater of the floor of the middle fossa was elevated and the gasserian ganglion was exposed. This structure was found to be displaced laterally by a mass which was present behind. The ganglion was incised and a round, reddish-brown tumor, 1.5 cm. in diameter, was noted. This tumor was removed as completely as possible. Neurologic examination postoperatively revealed complete motor and sensory loss of the 5th nerve.

The patient returned on Sept. 8, 1954, because of left occipital headaches of 2 weeks' duration. Results of neurologic examination were unchanged from those previously noted, except for the presence of a vestibulocerebellar type of nystagmus on gaze to the left. Hearing was normal bilaterally. Roentgenograms of the skull revealed no change from those previously noted. Observation for 2 months was advised.

The patient returned on Dec. 1, 1954, complaining of progressive loss of vision in the left eye during the previous 4 weeks, with slight inco-ordination of the left hand, and headaches as before. Neurologic examination revealed nystagmus, with complete motor and sensory loss of the 5th nerve. The ocular fundi were normal. Examination of the visual fields revealed a dense central scotoma of the left eye, breaking through to the inferior nasal quadrant, indicating a prechiasmal lesion of the left optic nerve. Roentgenologic examination of the optic canals and skull disclosed marked enlargement of the left optic canal, and increased decalcification at the tip of the left petrous pyramid.

On Dec. 7, 1954, ventriculography was carried out, but satisfactory filling of the ventricles was not obtained. The ventricular fluid contained 400 mg. of protein per 100 cc. Left transfrontal craniotomy was carried out on the same day, and it was found that the left carotid artery was displacing the optic nerve against the foramen. The optic nerve was decompressed.

The patient returned on April 15, 1955. The left eye at this time was completely blind. Four days before admission the patient had experienced a sudden severe occipital headache and tinnitus in both ears more marked on the left side. Ventriculography was repeated on April 19. The ventricular system had shifted to the right indicating the presence of an expanding mass beneath the anterior and temporal horns on the left side. On the same day left transtemporal craniectomy was carried out. Along the floor of the middle fossa a large neurofibroma (neurilemmoma) was found extending anteriorly into the region of the chiasm and posteriorly to involve the tip of the petrous process. Radical intracapsular enucleation was done, and as much of the capsule as possible was removed. That portion of the tumor that was removed was about 4 cm. in diameter.

Postoperatively, the patient had an uneventful convalescence. He was treated with emanations from a cobalt bomb. When he was last heard from on June 25, 1955, he appeared to be getting along very satisfactorily.

Case 3.—A white married woman 31 years old registered at this clinic on April 1, 1955. Three months previously, she had undergone extraction of four upper teeth, and dentures had been fitted. This dental work required about a month. Two weeks after the plates were in place, the patient first noticed numbness around the right cheek and along the right side of the nose. One week before her registration at the clinic, a dull, aching pain localized around the right eye was first noticed. This pain would come on suddenly and would persist for about 5 minutes, and it was relieved by rest and hot packs. During the 3 days prior to her registration, the patient

noticed some blurring in the right eye when she looked at distant objects and particularly when she looked to the right.

Neurologic examination revealed total anesthesia and analgesia over the 1st branch of the right trigeminal nerve, and moderate hypesthesia and hypalgesia over the 2nd and 3rd divisions. The corneal reflex was absent. The pterygoid, masseter and temporal muscles on the right side were noticeably weak, and there appeared to be some degree of atrophy of these muscles. Ophthalmologic examination revealed normal ocular fundi and visual fields. There was moderate weakness of the right lateral rectus muscle, with diplopia on gaze to the right. A small corneal ulcer was present in the right eye. Roentgenograms of the skull disclosed slight decalcification at the right foramen ovale. A diagnosis of a lesion of the right trigeminal nerve was made.

On April 29, 1955, a right carotid angiogram was made. A slight elevation of the middle cerebral artery was demonstrated. On May 2, right transtemporal craniotomy was carried out. While the dura mater was being reflected from the 3rd divi-

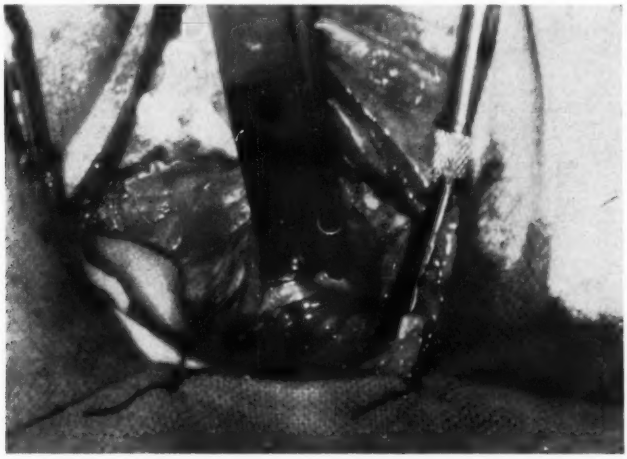

FIG. 5. *Case 3.* Neurofibroma arising from the right gasserian ganglion in the middle fossa.

sion of the trigeminal nerve and the inferior border of the gasserian ganglion, a bluish-yellow mass was noticed (Fig. 5). Additional exposure revealed the mass to be about 2 cm. in diameter. A portion of the mass was cystic. The cystic area was opened, and about 1 cc. of dark, liquid blood escaped. Biopsy of tissue from the tumor proved it to be a neurofibroma. The tumor appeared to have its origin from the gasserian ganglion. It extended backward along the posterior root for a short distance. All the tumor that could be visualized was removed. It was necessary to sacrifice both motor and sensory branches in order to effect as complete removal as possible of this tumor. Postoperative convalescence was uneventful. Subsequent neurologic examination showed complete right trigeminal motor and sensory deficits. There was some abatement of the lateral rectus palsy.

The rapid onset and progression of the symptoms in this case suggested metastatic involvement of the gasserian ganglion. However, the rapid onset and progres-

sion undoubtedly were explained by hemorrhage into the tumor, which appeared as a cystic area in the tumor.

Case 4.—A white married woman 55 years old registered at the clinic on Aug. 26, 1952. She said that since the autumn of 1949 she had experienced dull pain in front of and within the auditory canal on the right. By the summer of 1952 the dull pain had become worse, and was associated with intermittent, sharp, shooting pain. Since April, 1952, the shooting pain had become worse. It extended along the ramus of the right mandible.

Results of neurologic and otorhinolaryngologic examinations were essentially normal. A roentgenogram of the skull disclosed nothing abnormal. Roentgenograms of the spinal column showed narrowing of the interspace of the 5th cervical vertebra. It was thought that the pain originated from irritation of the 2nd and 3rd cervical nerve roots. Heat and cervical traction were advised and tried. The patient felt that these measures lessened the pain, to some extent.

The patient returned on March 18, 1953, saying that the pain had become worse. Neurologic examination disclosed slight weakness of the motor branch of the right trigeminal nerve; however, sensation in the trigeminal region was intact and the corneal reflexes were equal bilaterally. Diagnosis was deferred.

The patient returned on April 9, 1954, complaining of pain as before. Results of the neurologic examination were essentially unchanged. In retrospect, it can be seen that roentgenologic examination of the skull done at this time demonstrated sharp decalcification of the right petrous tip, medial to the internal acoustic meatus, and decalcification of the posterior wall of the right foramen ovale. On May 6 a nodular goiter was removed.

The patient returned on April 4, 1955, complaining that the pain was of the same character and in the same location as before, but was more severe and more constant. During the previous year she also had noticed that her neck was stiff. Neurologic examination disclosed moderate hypalgesia on the right side of the face, but tactile sensation was normal. The trigeminal weakness persisted. Horizontal nystagmus on gaze to the right was present. The ocular fundi and visual fields were normal. The nasopharynx was normal. Roentgenograms of the skull revealed the observations previously noted. The possibility of a chordoma was entertained, and repeated examinations were advised.

The patient registered again on July 15, 1955. The pain was more severe and constant, and it extended from the right ear down to the right paratracheal area. Examination revealed hypalgesia, more pronounced, of the 2nd and 3rd divisions of the trigeminal nerve to the right side of the face and tongue. Increased weakness of the masseter, pterygoid and temporal muscles on the right was noted. Hearing was normal. Roentgenograms of the skull disclosed additional erosion at the tip of the petrous process on the right side. A diagnosis of tumor of the right side of the middle fossa was made.

On July 29, right transtemporal craniectomy was carried out. The dura mater was elevated from the floor of the middle fossa, and as the 2nd branch of the trigeminal nerve was exposed, a grayish mass was seen. The mass was about 2.6 cm. in diameter; it appeared to be cystic. About 10 cc. of yellowish fluid was aspirated from it. Biopsy of tissue from the tumor revealed it to be a neurofibroma (neurilemmoma). Radical subtotal removal of the tumor was carried out. Neurologic examination in the postoperative period revealed no change in the findings that had been made previously.

Case 5.—A white married woman 33 years old registered at the clinic on July 29, 1949. Four years previously she had noted the sudden onset of deafness and tinnitus in the right ear. She had been examined by a local otorhinolaryngologist, who said the auditory canal was obstructed by a neoplasm protruding through the eardrum. During this time, when she was overexcited, she also had had episodes which she described as "fainting spells." In November, 1948, the patient had first noted pain in the region of the inner canthus of the right eye. At this time she also had noticed numbness of the right side of the tongue and the roof of the mouth. A specimen of tissue from the tumor protruding through the eardrum was taken for biopsy and roentgen therapy was given. Several months afterward the patient noticed the onset of numbness of the right arm and leg. She also noticed some difficulty in swallowing. She was examined in May, 1949, by a surgeon in the local area, who again took tissue from the tumor for biopsy and reported the tissue as being neurofibromatous. At this time roentgenograms of the skull revealed erosion of the apex of the right petrous process and the posterior part of the floor of the middle fossa.

Physical examination at the time of the admission of the patient to the clinic gave normal results. Neurologic examination disclosed nystagmus in all directions, diminished corneal reflex on the right, weakness of the right masseter, temporal and pterygoid muscles, diminished sensation in the right trigeminal area and fasciculations of the tongue. Palatine and pharyngeal reflexes were intact. The patient experienced some degree of ataxia on turning from the left to the right. The ocular fundi, pupillary reactions and visual fields were normal. A smooth, fibrous tumor was seen protruding from the lower two thirds of the right tympanic membrane. The caloric response on the left side was normal, but on the right side it was absent. Deafness was complete on the right side. Roentgenograms of the skull revealed erosion and destruction of the tip of the petrous portion of the right temporal bone and sclerosis of the mastoid cells on the right side. A preoperative diagnosis of tumor of the right side of the cerebellopontine angle (meningioma or neurofibroma) was made.

On August 6 right occipital craniectomy was carried out. The 5th, 8th, 9th, 10th, 11th and 12th cranial nerves on the right side were visualized and were seen to be normal. However, the 7th and 8th nerves appeared to be somewhat posteriorly displaced by what seemed to be a slight bulging behind them. It was also noted at exploration that the petrous portion of the temporal bone was eroded. A tumor was not noted in the cerebellopontine angle, and it was felt that the tumor originated within the petrous portion of the temporal bone, and that the tumor was not accessible from a posterior-fossa approach. Three 5-mg. radium needles were inserted behind the 7th and 8th nerves in the region of the bulging. During the surgical procedure there was a sudden decrease in the blood pressure, which was restored by the usual means.

The patient had a rather complicated postoperative course, but slowly recovered. During her convalescence it was noted that she had inferior altitudinal, congruous right and left homonymous hemianopsia, presumably caused by lesions in the upper portions of both occipital lobes, most likely the results of cerebral ischemia.

The patient was dismissed. She returned again on Nov. 14, 1955. At that time she said that during the preceding 10 months she had experienced severe, dull, aching frontal pain, worse when she was stooping over and when she coughed.

During the preceding 4 or 5 months the right side of the face had become "numb" in the region of the 2nd division of the right trigeminal nerve. The patient also complained of clumsiness and numbness of the right hand. Neurologic examination demonstrated marked right corneal hypalgesia and also marked hypesthesia in the right side of the face. Moderate weakness of the right masticatory muscles and right hemiatrophy of the tongue were recorded. Inco-ordination of the right extremities also was present. The ocular fundi were seen to be flat; the visual fields were unchanged from the appearance noted during the convalescence of the patient after the first operation. The right middle ear was found to be scarred, but a tumor was not seen. The nasopharynx was normal. Roentgenograms of the skull demonstrated extensive destruction of the floor of the middle fossa and sharp decalcification at the petrous apex on the right side. A diagnosis of recurrent tumor, neurofibroma, of the 5th nerve was made.

On Nov. 29, 1955, right transtemporal craniectomy was carried out after the right external carotid artery had been ligated. After the dura mater had been stripped from the floor of the middle fossa for a distance of about 1.5 cm. from the edge of the craniectomy wound, a grayish-brown tumor was exposed. Exposure was continued and a neurilemmoma about 6 by 4 by 4 cm. was found. The tumor extended over to the midline, backward to the petrous bone and forward to the sphenoid ridge. The tumor also had eroded through the floor of the middle fossa and extended down to the nasopharynx. Extensive intracapsular enucleation was carried out.

The patient's convalescence was uneventful. Neurologic examination on the 7th postoperative day revealed no added neurologic defect. The patient said that she had no additional pain, and she did not have ataxia. She was dismissed on the 9th postoperative day.

Case 6.—A white married woman 45 years old registered at the clinic on April 21, 1948. She said she had first noticed enlarged lymph nodes in the cervical region 2 years prior to her registration. The masses had increased gradually, and large nodes had developed in the axillas and groins. A diagnosis of Hodgkin's disease had been made elsewhere. Biopsy at the clinic revealed lymphosarcoma of the Hodgkin type. Roentgen irradiation and nitrogen mustard were given.

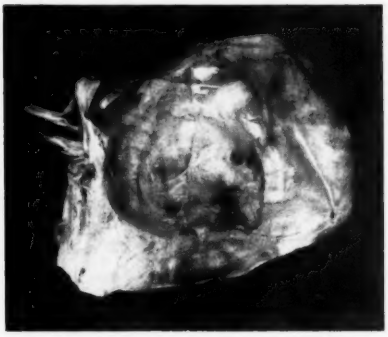

FIG. 6. *Case 6.* Neurofibroma of the left gasserian ganglion.

The patient returned 7 months later because of swelling of the feet, intermittent fever and a tendency toward bleeding. Culture of specimens of blood produced *Staphylococcus aureus.* The patient died 2 months after this second admission. During the period of clinical observation there had been no signs of involvement of cranial nerves.

At necropsy a firm tumor 2.5 by 2 cm. was found extending from the left gasserian ganglion. The mass incorporated the ganglion, and the foramen ovale was enlarged to about three times normal size. The tumor followed the mandibular division of the 5th nerve for a short distance. On microscopic

examination the tumor proved to be a neurofibroma (Figs. 6 and 7).

Summary on Supratentorial Tumors.—Tumors arising from the fifth nerve within the middle fossa, rather than conforming to a constant clinical pattern, often manifest themselves by symptoms suggesting locations other than the middle fossa. For example, in Case 1 the lesion presented as a chiasmal lesion; in Case 5 clear-cut symptoms of a tumor of the cerebellopontine angle were present.

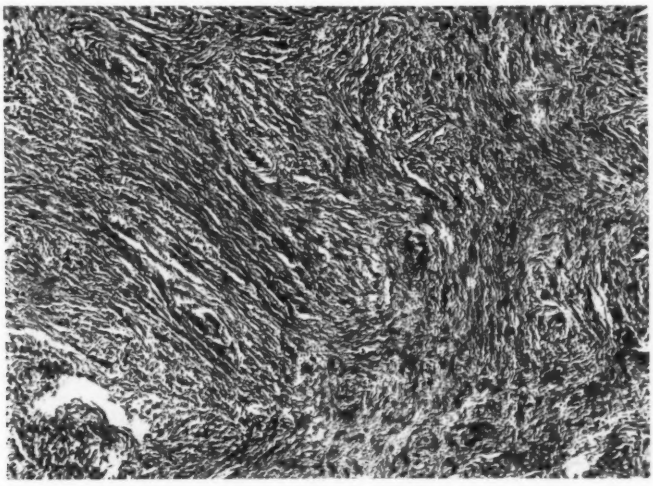

FIG. 7. *Case 6.* Neurofibroma (neurilemmoma) of left gasserian ganglion, showing fibrils of reticulin (reticulin impregnation, ×110).

In Case 2 symptoms clearly suggested a tumor of the middle fossa, and both the fifth and the ipsilateral optic nerve were involved. This suggestion was substantiated by roentgenologic findings.

In Case 3 symptoms referable to the fifth nerve predominated. The onset of facial paresthesias, very shortly followed by severe facial pain and the findings of facial analgesia and weakness of the masticatory muscles, pointed to involvement of the middle fossa.

Pain within the ear was the initial complaint in Case 4. Later, clear-cut involvement of the fifth nerve and roentgenologic changes pointed to the lesion in the middle fossa.

Case 6 is an interesting one because of the fact that a tumor 2.5 by 2.5 by 2 cm. was present in the gasserian ganglion without any symptoms or signs of involvement of the middle fossa.

SUPRATENTORIAL AND INFRATENTORIAL TUMORS

Case 7.—A white man 32 years old first registered at the clinic on July 22, 1952. He had first noticed tinnitus in the right ear, with progressive deafness, 4 months previously. In the 5 months before his registration he had also noted some blurring of vision.

Ophthalmologic examination revealed bilaterally chronically choked disks of 4 diopters' elevation, with early pallor. Examination of the visual fields revealed lower nasal contraction and an upper temporal scotoma in the right visual field, and a centrocecal scotoma and upper nasal contraction in the left visual field. A slightly ataxic gait, increased tendon reflexes bilaterally and left extensor plantar reflex were recorded. Roentgenograms of the skull revealed marked destruction of the middle fossa, and erosion of the petrous tip on the right. The right anterior clinoid process appeared to be elevated and eroded. Electroencephalographic study disclosed grade 2 generalized dysrhythmia, maximal in the bitemporal regions, but stronger on the right side. A tentative diagnosis of petrosphenoid tumor (possibly chordoma) was made.

Ventriculography was carried out on July 25, 1952. It revealed dilatation of the lateral and third ventricles. The fourth ventricle and aqueduct were not visualized. The protein content of the ventricular fluid was 2,000 mg. per 100 cc. On the same day right transfrontal craniotomy was carried out. A cystic mass was found beneath the right internal carotid artery in the right half of the visual apparatus. The cyst was punctured and about 20 cc. of xanthochromic fluid were obtained. The convalescence was uneventful.

The patient returned to the clinic on September 16 of the same year. During the interim since dismissal he had noticed marked decrease in hearing and intermittent, dull, frontal headaches.

Examination disclosed partial loss of hearing on the right side, bilaterally choked disks of 3 diopters' elevation and slightly ataxic gait. Roentgenograms of the skull revealed no change from the previous appearance. A diagnosis of tumor of the right side of the cerebellopontine angle was made.

On September 26 right suboccipital craniectomy was carried out. High up in the cerebellopontine angle a mass presented, which on biopsy proved to be a neurofibroma. The tumor was removed almost completely, as far as it could be visualized. The tumor was not attached to the 8th intracranial nerve at any point. The posterior root of the 5th nerve was intimately associated with the tumor, and it was the opinion of the surgeon that the tumor had originated from the 5th nerve. The tumor weighed 10 gm. and was about 3 cm. in diameter. The patient had an uneventful convalescence. Neurologic examination, after this second operation, disclosed complete right trigeminal anesthesia and analgesia. The corneal reflex was absent on the right side. The patient was not seen for re-examination after dismissal.

Case 8.—A white married man 38 years old registered at the clinic on Aug. 30, 1946. He said that for the previous 16 years he had noticed occasional bilateral frontal headaches, accompanied by nausea and vomiting. These headaches had occurred on the average of two to three times a year, and persisted for 24 to 48 hours. During the year prior to his registration, he had noticed that headaches in the left temporal region would arise in the early morning and would increase on sudden motion of the head and become progressively more severe and frequent. The patient also had noted, in the few months prior to his registration, double vision on looking to the left. Six months before, he had noticed intermittent numbness of the right

side of the face, limited to the forehead and region of the cheek, and occurring six or seven times daily.

Neurologic examination demonstrated moderate paresis of the left external rectus muscle, with convergent strabismus. The left corneal reflex was slightly diminished, but no other neurologic observations were made. Roentgenograms of the skull disclosed destruction of the floor of the middle fossa in the region of the foramen ovale and the tip of the petrous pyramid on the left side.

A diagnosis of tumor of the middle fossa was made. On September 4 left temporal craniotomy was carried out. A tumor could not be found until the posterior root of the trigeminal nerve had been sectioned and reflected. A mass was then evident; it arose from this root and was about 1.5 cm. in diameter. Biopsy proved it to be a neurofibroma. It was thought that the entire tumor was removed. Convalescence was uneventful. Neurologic examination revealed numbness of the left half of the face, together with 5th-nerve motor paresis on the left side.

The patient returned on Aug. 21, 1950. During the year prior to this registration he had experienced left occipital and temporal headache. He again returned for examination 13 months later, in September, 1951, complaining of progressive ataxic gait of 3 months' duration. Neurologic examination revealed the previous defects and marked ataxia of the lower extremities. Hearing was normal, but marked hypofunction of the left vestibular apparatus was noted. Roentgenograms of the skull revealed no further changes.

On Nov. 15, 1951, the left transtemporal incision was reopened and the middle fossa was inspected. A tumor was not present in the middle fossa. The tentorium was then split, and a tumor 3 cm. in diameter was seen. It was situated in juxtaposition to the left cerebral peduncle. Total radical removal of the tumor was carried out. The patient's convalescence was somewhat stormy, complicated by agraphia and alexia, during the first 2 weeks. Re-examination carried out 17 months later revealed some degree of residual agraphia and alexia, complete sensory and motor impairment of the left 5th nerve and moderate ataxia.

Case 9.—A white married man 31 years old registered at the clinic on Aug. 12, 1949. He said that about 5 months previously he had first noticed that headache would start daily in the neck and extend over the forehead, aggravated by effort. About the same time he noticed a sensation that the right ear was "stuffed up," and he experienced episodes of dizziness which appeared intermittently, associated with tinnitus. About 3 months prior to registration, he had first noticed numbness of the right side of the face which progressed from the forehead to include the nose and the lower jaw, over a 2 months' period. He also had noticed the gradual onset of loss of taste in the right side of his tongue, and also difficulty in walking. About 6 months before he came to the clinic he had begun to experience intermittent vomiting. His left hand and fingers became numb and clumsy, and he also began to hiccup periodically.

The ocular fundi were normal. Neurologic examination disclosed a vestibulocerebellar type of nystagmus, absence of the right corneal reflex, hypesthesia in the distribution of the 3rd branch of the right trigeminal nerve, slight weakness of the right masseter and pterygoid muscles on the right, and weakness of the right orbicularis oculi muscle. The gait was ataxic and Romberg's sign was present. Audiometry demonstrated moderate conduction deafness. The right labyrinth was hypoactive on caloric stimulation. Roentgenograms of the skull disclosed no abnormality. A diagnosis of tumor of the cerebellopontine angle on the right was made.

On Aug. 17, 1949, right suboccipital craniectomy was carried out. A tumor, which biopsy proved to be a neurofibroma, was found high in the cerebellopontine angle. The tumor was completely removed, and the 5th nerve was exposed. The 8th nerve appeared to be intact. The tumor was 3 cm. in diameter. Convalescence was uneventful. Neurologic re-examination at the time of dismissal disclosed complete sensory and motor deficit of the right trigeminal nerve, and absence of taste and sensation on the right side of the tongue. There was no further impairment of hearing.

The patient returned to the clinic on March 21, 1953. He complained of paresthesia of the left hand and weakness of the left leg. Neurologic examination revealed the gait to be moderately ataxic, and tendon reflexes were increased on the left side, with extensor plantar response on the left. The findings in respect to the 5th, 7th and 8th cranial nerves were unchanged. The optic disks were flat. Roentgenograms of the skull revealed no abnormality.

On June 9, 1953, right temporal craniectomy was done. An extradural tumor on the floor of the middle fossa was found; it had invaded the 2nd and 3rd divisions of the 5th nerve and the ganglion. The tumor was about 2.5 cm. in diameter. A small portion of the tumor extended through the tentorial notch into the posterior fossa. At the time of his dismissal, the patient was improving.

Summary on Supratentorial and Infratentorial Tumor.—In Cases 7 and 9 symptoms and signs referable to the posterior fossa predominated, and suggested the tentative diagnosis of chordoma in the former case and tumor of cerebellopontine angle in the latter. From the surgical standpoint, it is particularly important to be aware of the possibility of hour-glass tumor when trigeminal neurofibromas are at hand. If it is known that the tumor invades both the supratentorial and infratentorial regions, the temporal approach and sectioning of the tentorium are indicated: this is illustrated in Case 8 of the present series.

INFRATENTORIAL TUMORS

Case 10.—A white married woman 45 years old registered at the clinic on May 17, 1944. She said that 3 years previously she had noticed the sudden onset of anesthesia of the right side of the face, followed very shortly by right facial weakness. She also complained of generalized headache and intermittent tinnitus on the right side. During the 4 months prior to her registration, staggering gait and dizziness were present. A month before her registration the inco-ordination became more evident, as did the facial weakness. The patient also noticed that the taste on the right side of her tongue was impaired. A corneal ulcer and impaired hearing, both on the right side, had developed within the last few weeks prior to her registration.

A large corneal ulcer was found on the right eye, and corneal anesthesia was complete. Moderate nystagmus was elicited on lateral gaze to the right. Loss of sensation was complete in the distribution of the right trigeminal nerve, and marked weakness of the masseter, pterygoid and temporal muscles was recorded. Facial palsy on the right was almost complete. Inco-ordination of the right side of the body was present, together with ataxic gait. The ocular fundi and the visual fields were normal. Labyrinth response on the right was absent. Roentgenograms of the skull disclosed essentially nothing abnormal. A diagnosis of tumor of the cerebellopontine angle (possibly meningioma) was made.

On May 24 right suboccipital craniectomy was carried out. A mass was found

high up in the cerebellopontine angle. Biopsy of tissue from the tumor proved it to be a neurilemmoma. It was about 2.5 cm. in diameter and was partially cystic. It appeared to originate from the 5th cranial nerve. The tumor was removed subtotally. The 7th, 8th, 9th and 10th cranial nerves were identified after the tumor had been removed; they appeared to be intact. Convalescence was uneventful. Neurologic re-examination at the time of dismissal of the patient showed her to be free of headache, dizziness and tinnitus. There appeared to be some improvement in the sensation of the right trigeminal area. Hearing remained absent on the right side. Ataxia was abating steadily.

The patient returned on July 22, 1945, because of recurrence of unsteady gait, associated with weakness of the left arm and leg, slurred speech and regurgitation of fluids during the 6 months prior to this registration. The symptoms had become markedly pronounced during the previous 4 weeks.

The speech was markedly explosive. Coarse nystagmus on gaze to the right side was noted and some degree of peripheral weakness of the right facial nerve, analgesia of the right side of the face, absence of the right corneal reflex, moderate weakness of the left extremities, with bilateral plantar extensor responses, and marked incoordination of all four extremities were recorded. Funduscopic examination revealed no evidence of choked optic disks. A diagnosis of recurrent neurofibroma was made, but operation was declined. A course of roentgen therapy was advised and the patient was dismissed. The patient died in November, 1946. Necropsy was not done.

Case 11.—A white married man 44 years old registered at the clinic on Oct. 24, 1941. He said that during the preceding 2 months he had noticed, over the left eye, a continuing numb feeling which sometimes involved the left cheek, the left side of the tongue and the left corner of the mouth. During the same period he also had noticed some degree of diplopia on looking to the left.

Horizontal nystagmus, moderate weakness of the left lateral rectus muscle, and diminution of the left corneal reflex were found. Roentgenograms of the skull showed nothing abnormal. No definite diagnosis was made. The patient was advised to return in 3 months.

In March, 1943, the patient returned, reporting that the numbness of the face had become more extensive, and involved the left half of the face, the mouth and tongue. During the previous 6 months, he had noticed tinnitus in the left ear and some ataxia, and tendency to fall to the right. Diplopia had become more frequent and more noticeable. During the 3 months prior to this registration, the patient had noticed difficulty in swallowing and also slurred speech.

Neurologic examination disclosed analgesia and paresis of the left trigeminal motor root, diminution of the left corneal reflex, slight paresis of the left facial nerve and absence of taste on the left anterior portion of the tongue. The left external rectus muscle was weak. Speech was markedly slurred. Moderate dysmetria was noted on the left extremities, and the gait was ataxic. Tendon reflexes on the left side were hyperactive. A mild, high hearing loss was recorded bilaterally, and the left labyrinth was hyperactive. The ocular fundi were normal. Roentgenograms of the skull disclosed erosion of the left petrous tip and the floor of the sella turcica. A diagnosis of tumor of the left side of the cerebellopontine angle was made; it was thought that the lesion was either a meningioma or an acoustic neurofibroma.

On March 19, 1943, left suboccipital craniectomy was carried out. A tumor, which biopsy proved to be a neurofibroma (neurilemmoma), was found high in the left cerebellopontine angle. The tumor was cystic, and was about 3.5 cm. in diameter.

It was situated adjacent to the tentorium, and appeared to arise from the 5th nerve. The tumor displaced the pons and brain stem mesially, and extended downward and displaced the 7th and 8th nerves. Intracapsular enucleation was done. The patient's convalescence was satisfactory until the 5th postoperative day, at which time his condition became suddenly worse. He died on the 8th postoperative day. Necropsy was not performed.

Case 12.—A white married man 34 years old registered at the clinic on Oct. 1, 1946. He had been well until June, 1945, at which time he had noticed numbness in the right side of the upper lip, which gradually extended to encircle the eyes and also to include the upper and lower jaws. About the same time he had noticed generalized, dull, aching headaches on awakening. These would persist for about an hour. In November, 1945, he had noticed that his gait was unsteady and that he had a tendency to fall to the right. At this time also he had noticed a tingling of the fingers of the left hand and numbness of the left arm. Episodes of nausea and vomiting occurred, and swallowing became difficult. Ventriculography and suboccipital craniectomy had been carried out elsewhere, but they did not disclose a tumor. A course of roentgen therapy was prescribed for a suspected infiltrating glioma of the pons. The patient's symptoms had persisted, however.

When this patient was first seen at the clinic, neurologic examination disclosed a somewhat ataxic gait, mild horizontal nystagmus, slight hypesthesia over the right side of the upper lip, diminution of the right corneal reflex and slight weakness of the right side of the tongue. A roentgenogram of the skull gave evidence of a suboccipital craniectomy and bilateral trephine openings. Observation was advised, with the request that the patient return in 5 or 6 months.

The patient did not return until June 28, 1950. He had noticed increased unsteadiness of gait and return of the nausea and vomiting. He also had noticed right-sided deafness. Neurologic examination disclosed hypesthesia and hypalgesia of the 1st and 2nd divisions of the right trigeminal nerve, moderate weakness of the right masseter, temporal and pterygoid muscles, and a marked ataxic gait. Hearing was moderately impaired on the right side.

The patient returned again 4 months later. The numbness on the right side of his face had increased, and he was aware of increased ataxia. Neurologic examination disclosed rather marked ataxia, hypalgesia in all three divisions of the right trigeminal nerve, and nystagmus in all directions. Neurologic observations otherwise were the same as those made in previous examinations. The ocular fundi were normal. Roentgenograms demonstrated localized erosion of the apex and of the right petrous bone, and decalcification of the foramen ovale on the same side. The pineal gland was observed to be displaced to the left. A diagnosis of meningioma or neurofibroma in the region of the petrous tip was made, and operation was advised.

On Nov. 2, 1950, right temporo-occipital craniectomy was done, and the middle fossa was explored. The tentorium was bulging upward. It was split surgically, and a tumor was noted underneath the tentorium, extending into the right cerebellar lobe; it was 2.5 cm. in diameter. The tumor arose from the posterior root of the trigeminal nerve. Biopsy proved it to be a neurofibroma (neurilemmoma). The tumor was completely removed.

Postoperative convalescence was uneventful, Neurologic re-examination showed improvement, as far as ataxia and nystagmus were concerned. Complete analgesia and anesthesia were noted in the distribution of the right 5th nerve.

Case 13.—A white man 21 years old registered at the clinic on Jan. 12, 1955. His father said the patient had been alert and in good health until 1951, at which time

it had been noticed that the patient laughed readily without provocation. Attacks of severe frontal headache had begun about the same time; they would persist for about 2 or 3 hours and would occur every 2 or 3 weeks. Two years before his registration the patient had noticed gradually progressive weakness of the left extremities. Since July, 1954, slurred speech, weakness and a sensation of "stiffness" of the left side of the face and difficulty in swallowing, with occasional regurgitation of fluids through the nose, had been present.

Results of the general physical examination were essentially negative. Neurologic examination revealed a vestibulocerebellar type of nystagmus, maximal when looking to the left, a peripheral type of weakness of the left facial nerve, slight diminution of the left corneal reflex, moderate weakness of the left side of the soft palate, presence of the sucking reflex, moderate weakness of the left extremities, absence of abdominal reflexes, bilateral presence of extensor plantar reflexes, diminished two-point sensation over the left hand and marked inco-ordination bilaterally. The patient was somewhat euphoric, and laughed at the slightest provocation. Roentgenograms of the skull disclosed asymmetry of the petrous tips. Electroencephalography disclosed grade 1 generalized dysrhythmia.

A diagnosis of pseudobulbar palsy or pontine tumor was made, and pneumoencephalography was carried out on Jan. 17, 1955. These studies revealed the aqueduct and fourth ventricle to be displaced posteriorly and to the right. The total protein content of the cerebrospinal fluid was 600 mg. per 100 cc. On Jan. 19, left suboccipital craniectomy was performed, and a large bluish-gray mass was found high up in the left cerebellopontine angle. Biopsy proved it to be a neurofibroma (neurilemmoma). Intracapsular enucleation was carried out, and as the operation proceeded the tumor was noted to lie anterior to the pons. The tumor arose from the 5th nerve and projected up through the tentorial notch and extended downward to a level 1.5 cm. below the internal acoustic meatus. The tumor measured 3.5 by 4 cm. and weighed 18.2 gm. Except for a small part of the capsule, the tumor was completely removed.

Postoperative convalescence was uneventful. At the time of dismissal the patient had improved in all respects, except that he had analgesia in the distribution of the left trigeminal nerve. The pharyngeal reflex was slightly diminished, but the patient was able to swallow well. He no longer had episodes of unprovoked laughter, and he was mentally alert and oriented.

Summary on Infratentorial Tumors.—In all of the 4 preceding cases, the symptoms and signs pointed to involvement of the posterior fossa by a tumor. In 3 of these cases the pattern was similar. Signs of involvement of the fifth nerve were followed by findings referable to involvement of the eighth nerve and cerebellar involvement. In Case 13, the symptoms and signs suggested pseudobulbar palsy, but were predominantly unilateral and the roentgenograms of the skull, together with pneumoencephalograms, pointed to the situation of the tumor. Case 12 has been reported previously by Baker and Bailey,[5] who have already pointed out the importance of roentgenologic studies in early and accurate diagnosis.

COMMENT

Symptoms and Signs Referable to the Fifth Nerve.—The striking finding in this review is the fact that in 3 instances (Cases 1, 6 and 7) no objective in-

volvement of the fifth nerve was present. In Cases 1 and 7 the tumor was large, measuring 6 by 3.5 cm. in Case 1, and 3 cm. in diameter in Case 7. In Case 6 the tumor was smaller, being 2.5 by 2 cm. In Cases 1 and 6 the tumor was limited to the supratentorial space. In the remaining 10 cases, signs referable to the fifth nerve were present; however, in only 6 cases (2, 3, 4, 10, 11 and 12) did they constitute the first symptom. In 3 cases (5, 9 and 13) some change in the cutaneous sensibility of the face was present, but this change was not the first symptom noted. In 1 case (8) diminution of the corneal reflex constituted the only sign referable to trigeminal-nerve impairment.

The duration of the changes in facial sensibility, expressed as "numbness" by the patients, varied between 3 years and 6 months. In Cases 2, 11 and 12 these changes started in the first or second divisions and gradually extended to cover the whole face. In Case 12 serial neurologic examinations revealed hypesthesia of the second division at the time of the first examination, of the second and first at the time of the subsequent admission, and complete anesthesia of all divisions at the time of the last admission.

In Case 3 a "numb feeling" developed in the second division; the next week the patient had the sensation that her face "was going to sleep." Subsequently, severe pain developed in the same place. Examination revealed total anesthesia and analgesia of the first division, and moderate hypalgesia and hypesthesia of the second division.

In 8 cases, variable degrees of weakness of the masticatory muscles were demonstrated.

It is interesting to note that in 2 cases signs of involvement of the fifth nerve developed rapidly. In Case 10 the onset of alteration in facial sensibility was almost sudden, associated with the development of facial weakness. The latter possibly had a basis in traction exerted upon the greater petrosal superficial nerve. In Case 3 paresthesias, originally detected by the patient in the second division, involved all three divisions within a week. In this particular case, a hematoma of recent origin was found in the tumor at operation. In the former case a cyst was present in the tumor, but no mention of recent hemorrhage was made by the surgeon in his operative note.

Symptoms and Signs Referable to the Eighth Nerve.—In 9 cases the tumor lay in juxtaposition to the eighth nerve. In 4 of these cases the tumor was situated entirely in the posterior fossa; in 3 cases the tumor was both supratentorial and infratentorial, more or less equally distributed; finally, in 2 cases the main bulk of the tumor was situated supratentorially, with a smaller extension into the infratentorial space.

In 8 of all 13 cases (2, 5, 7, 8, 9, 10, 11 and 12) there were signs of involvement of the eighth nerve. In Case 13, in which a tumor of the posterior fossa was found, there was no sign at all of impairment of the eighth nerve.

In 2 cases (5 and 7), symptoms of involvement of the eighth nerve constituted the initial complaint, and in both cases the surgeons' operative notes did not mention any attachment of the tumor to the acoustic nerve.

Impairment of the vestibular branch was evident in 5 cases (2, 5, 8, 9 and

11); in 2 of them this type of impairment was associated with objective deafness; in another 2 it was associated with tinnitus; and in 1 case impairment of the vestibular branch constituted the only sign of impairment of the eighth nerve.

Objective deafness was present in 5 cases (5, 7, 9, 10 and 12). In 4 of them nerve deafness was most likely, and in 1 the presence of conductive deafness was demonstrated.

In 2 cases tinnitus without objective deafness was present.

Involvement of Other Cranial Nerves.—In 9 cases of this series the tumor, or a portion of it, occupied the middle fossa, and thus lay near the optic pathways. In 3 of these 9 cases evidence of involvement of the optic pathways was obtained. In 1 of these 3 cases no objective evidence of involvement of the fifth nerve was present, and this fact resulted in a misleading localization: most of the tumor was missed during operation (Fig. 3). In another case evidence of unilateral involvement of the optic nerve developed some time after partial removal of the tumor. This impairment was associated with changes in the corresponding optic canal. Subsequent exploration in this case demonstrated notching of the optic nerve produced by compression of this structure between the carotid artery and the optic canal. In the third case, a nonlocalizing type of field was obtained, and ventriculography demonstrated enlargement of the third ventricle. It should be pointed out that in Case 5 bilateral homonymous hemianopsia occurred immediately after the first surgical operation, most likely the result of vascular changes in the occipital lobe.

Papilledema was present only in Case 7.

Only the sixth nerve showed evidence of impairment, as far as the motor nerve of the eye is concerned. This confirms previous observations[19] that these nerves are affected infrequently by neurofibromas of the fifth nerve. The sixth nerve was affected in 5 cases in the present series.

Signs of involvement of the seventh nerve were present in 5 instances (Cases 2, 9, 10, 11 and 13). In 3 of them the bulk of the tumor was situated in the posterior fossa. In Case 2 most of the tumor occupied the middle fossa; however, at operation a small projection into the posterior fossa was seen. In Case 9 the tumor was situated in both fossas.

Symptoms referable to involvement of the tenth nerve were present in 3 cases (5, 11, and 13). In 2 of them the tumor was situated infratentorially. Only in Case 13 was objective evidence of deficit of the tenth nerve found.

Paresis of the tongue was present in Cases 5 and 12. In the former the tumor had eroded the floor of the middle fossa and extended in the soft tissues at the base of the skull. In the latter the tumor was situated in the posterior fossa.

Other Signs.—It has long been recognized[14,21] that ataxia is present when neurofibromas arise from the posterior root of the trigeminal nerve. We observed that ataxia was a very constant sign in the present cases, when the tumor was situated infratentorially. Evidence of ataxic gait and inco-

ordination of the extremities was observed in all 7 cases of involvement of the posterior fossa, and also was present in 2 cases in which the tumor lay predominantly in the middle fossa.

Ipsilateral hemiparesis of the extremities was present in Case 13, in which the tumor was limited to the posterior fossa. The hemiparesis evidently was produced by displacement of the brain and the production of a notching of the cerebral peduncle against the tentorium. This observation also has been made occasionally in the presence of tumors of the eighth nerve.[20]

Determinations of the protein content of the cerebrospinal fluid were carried out in Cases 12 and 13. Protein amounted to 80 mg. per 100 cc. in the former and 600 mg. per 100 cc. in the latter. In Cases 2 and 7, determinations of the protein content of the ventricular fluid were 400 mg. and 200 mg., respectively, per 100 cc. of fluid. These findings agree with similar observations made in the presence of acoustic neurofibromas, and are helpful in diagnostic identification of the tumor.

PATHOLOGIC OBSERVATIONS

Study of the gross specimens removed at operation and observations at postmortem examination in Case 1 evidenced the frequency with which cystic degeneration occurs in these tumors. It should be mentioned that in Case 7 a cystic cavity was found in the middle fossa during the first exploration, and subsequent posterior-fossa craniectomy disclosed a more solid mass situated infratentorially. Similar findings have been reported previously.[13,19]

Microscopically,[25] zones of palisading arrangements of fibrils and cells were observed in the present series; however, apolar arrangement with microcystic degeneration was found more frequently. Impregnation with silver stains disclosed reticulin in all but degenerated areas.

SUMMARY AND CONCLUSIONS

Review of the literature has yielded a total of 43 cases of proved and accepted neurofibromas of the fifth nerve.

We have described the findings in 13 cases of solitary trigeminal neurofibromas (neurilemmomas) found at the Mayo Clinic. The tumors herein reported upon were divided in three groups for study. In 6 cases the tumors were restricted primarily to the middle fossa; in 4, to the posterior fossa, and in 3, the tumors were situated supratentorially and infratentorially.

Because of the misleading symptoms and signs frequently manifested during initial observation of the patients, it was difficult in some cases to make an early diagnosis. In many cases roentgenologic data were very helpful.

As to clinical findings in the presence of tumors of the fifth nerve, it was found that in 3 instances no objective involvement of the trigeminal nerve was present. In the remaining cases symptoms and signs consisted of some impairment of facial sensation, weakness of the masticatory muscles or facial pain.

In 8 instances signs of involvement of the eighth nerve were present.

The optic pathways were found to be affected in 4 instances. Papilledema occurred only once in the present series.

The sixth nerve was clinically impaired in 5 instances. The seventh nerve was impaired in 6. Symptoms of involvement of the tenth nerve occurred in 3 cases. Signs of deficit of the twelfth nerve were present in 2 instances.

Ataxia was a constant accompaniment in those cases in which the tumor was situated infratentorially.

Ipsilateral hemiparesis was found in 1 instance, suggesting notching of the midbrain against the tentorium.

The protein content was found to be increased in all cases in which the cerebrospinal or ventricular fluid was analyzed.

Pathologically, cystic degeneration was a common feature, and in all cases the tissue was identified as corresponding to neurofibroma (neurilemmoma) by the use of routine and special stains.

REFERENCES

1. Alajouanine, T., de Martel, T., and Guillaume, J. Schwannome du trijumeau rétrogassérien. *Rev. neurol.*, 1930, *2:* 89–94.
2. Alajouanine, T., Thurel, R., and Guillaume, J. Un nouveau cas de schwannome du trijumeau rétrogassérien. Ablation. Guérison. *Rev. neurol.*, 1935, *63:* 897–900.
3. Altmann, F. Zur Kenntnis der primären Geschwülste des Trigeminus und des Ganglion Gasseri. *Beitr. path. Anat.*, 1928, *80:* 361–404.
4. Auersperg, A. Ein Fall von Neurinom des N. trigeminus. *Nervenarzt*, 1937, *10:* 341–347.
5. Baker, G. S., and Bailey, A. A. Neurofibroma of the trigeminal nerve root in the posterior fossa: total removal by transtentorial approach; report of case. *Proc. Mayo Clin.*, 1952, *27:* 118–120.
6. Catalano, L. Su due casi di tumori della regione petroso-gasseriana. *Clin. nuova*, 1951, *12:* 547–555.
7. Chang, C. Surgical treatment of trigeminal neuralgia and description of a case of tumour of the Gasserian ganglion successfully removed in the Moukden Medical College Hospital. *Chin. med. J.*, 1935, *49:* 412–421.
8. Cohen, I. Tumors involving the Gasserian ganglion. *J. nerv. ment. Dis.*, 1933, *78:* 492–499.
9. Cushing, H., and Eisenhardt, L. Meningiomas. Their classification, regional behaviour, life history, and surgical end results. *Springfield, Ill.: Charles C Thomas*, 1938, xiv, 785 pp. (see pp. 208–223.
10. Davidoff, L. M., and Epstein, B. S. The abnormal pneumoencephalogram. *Philadelphia: Lea & Febiger*, 1950, 506 pp. (see pp. 108–111).
11. Dew, H. R. Some clinical syndromes of the fifth cranial nerve. *Aust. N.Z. J. Surg.*, 1933, *3:* 130–148.
12. Gaál, A. Zur Röntgendiagnose des Neurinoma Trigemini. *Röntgenpraxis*, 1935, *7:* 546–550.
13. Gjertz, E., and Hellerström, S. Tumeur du ganglion de Gasser. *Acta med. scand.*, 1925, *63:* 7–23.
14. Glaser, M. A. Tumors arising from the sensory root of the trigeminal nerve in the posterior fossa. Perineurial fibroblastoma. *Ann. Surg.*, 1935, *101:* 146–155.
15. Gonzalez Revilla, A. Neurinomas of the cerebellopontile recess. A clinical study of one hundred and sixty cases including operative mortality and end results. *Johns Hopk. Hosp. Bull.*, 1947, *80:* 254–296.
16. Graf, K. Cited by Loew and Tönnis.[23]
17. Guillaume, J., Rogé, R., and Mazars, G. Un cas de neurinome géant du ganglion de Gasser. *Rev. neurol.*, 1949, *81:* 225–226.
18. Hellsten, M. Ein Fall von Ganglion Gasseri-Tumor. *Dtsch. Z. Nervenheilk.*, 1914, *52:* 290–305.
19. Jefferson, G. The trigeminal neurinomas with some remarks on malignant invasion of the Gasserian ganglion. *Proc. Congr. neurol. Surg.*, 1955, *1:* 11–54.
20. Kernohan, J. W. Effects of displacement of the brain by tumors. *Arch. Neurol. Psychiat., Chicago*, 1949, *62:* 867–868.

21. Krayenbühl, H. Primary tumours of the root of the fifth cranial nerve: their distinction from tumours of the Gasserian ganglion. *Brain*, 1936, *59:* 337–352.
22. Lindgren, E. Das Röntgenbild bei Tumoren des Ganglion Gasseri. *Acta chir. scand.*, 1941, *85:* 181–194.
23. Loew, F., and Tönnis, W. Klinik und Behandlung der Neurinome des Nervus trigeminus. *Zbl. Neurochir.*, 1954, *14:* 32–41.
24. Nowotny, K., and Uiberall, H. Zur Kenntnis der Neurinome des Trigeminus. *Z. ges. Neurol. Psychiat.*, 1934, *150:* 75–99.
25. Olive, I. Intracranial neurilemmomas other than eighth nerve tumors. (Thesis) *Rochester, Minn.: Graduate School, Univ. Minn.*, 1956.
26. Orlando, R. Neurofibroma del trigémino. *Rev. neurol. B. Aires*, 1937, *1:* 445–451.
27. Shutova, T. A. Cited by Loew and Tönnis.[23]
28. Söderbergh, G. Ein Fall von Trigeminustumor mit Symptomen vom Kleinhirnbrückenwinkel, nebst einigen Bemerkungen über die sogenannte cerebellare Ataxie. *Nord. med. Ark.*, 1909, *42* [pt. 2 (*Ark. inre Med.*), No. 11]: 1–15.

DISCUSSION

Dr. James L. Poppen: I am sure that this is quite a change of pace from the discussion we had this morning with the chemopallidectomy symposium. I do want to compliment Dr. Svien on giving a factual detailed résumé of the 13 cases with which he has had experience.

When Dr. Svien first asked me to discuss the paper (which, by the way, you all want to read because of its excellence), and I saw 13 patients having a neurofibroma of the 5th cranial nerve, I thought immediately, "It can't be true. They must have many more."

However, I looked up our cases and excluded all the von Recklinghausen patients, and I soon noticed that it is a relatively rare condition. We had 9 patients who had solitary lesions involving the 5th cranial nerve. We did not have the three types. We had none solely in the infratentorial region. Ours were of the dumbbell type, of the middle fossa and involving the orbital fossa.

It would be folly to go ahead and give a résumé of our 9 patients, for they are mirror images of the cases he described, with the exception that we were not fortunate enough in having the smaller tumors. We had 4 patients who had marked exophthalmos with a tumor that extended into the middle fossa, and in one patient it displaced the pons posteriorly, wrapping itself around the basilar artery.

I do want to compliment Dr. Svien on the operative mortality. There, again, we were not quite as fortunate. We had 2 deaths directly ascribable to the operation. In one patient the basilar artery was opened and in the other the internal carotid had to be sacrificed. I feel that we ought perhaps to classify them as follows: the tumors that actually involve the sensory root, the type that involves the gasserian ganglion and those that are intrinsic in the nerve trunks distal to the ganglion. I think it is important in a way to know that in none of the series that I have read was the 3rd or the mandibular division peripherally involved with neurinoma. Having said this, I am sure several of you have had many of these cases. Neurinomas of the 5th cranial nerve are rare but either we do not have the inclination or we do not have the time to write up some of the rare conditions. For that reason, I am sure if a census were held of this group at this time that perhaps 20 to 40 more cases would come to view.

This morning six points were discussed to take care of this situation; but some of us still have the task of taking out brain tumors. Unfortunately, I cannot think of six points that I can give in order to have the perfect operation for neurinomas of the 5th cranial nerve.

In the first place, I could show you some patients who had marked exophthalmos from a neurinoma and afterward showed the eye in a normal position, but you cannot demonstrate by slides that a patient might be partially blind in one eye or that paresthesias of the face may develop after the removal of the tumor.

I do want just to mention the fact, as Dr. Svien has intimated, that the surgical approach is very important. When the tumor is predominantly in the posterior fossa one should attack

the tumor in the cerebellar angle and then incise the tentorium. Usually the tumor is not attached above the tentorium. On the other hand, if it arises in the region of the middle fossa, it is, of course, better to make a low temporal approach and incise the tentorium, as Dr. Svien stated, to be sure that the cerebellar angle is free from tumor.

If, however, the patient has an exophthalmos with an extension into the middle fossa, then the frontal temporal approach is preferable. I do want to warn you that in the 2 patients who died, the tumor arose in the region of the gasserian ganglion, and perhaps it is better, but completely against my philosophy, to leave a bit of the tumor behind. The reason I say that is when the patient has hypopituitarism, some visual field changes and perhaps 3rd nerve involvement, the tumor will be wrapped around the internal carotid artery. As happened in removing the last bit of tumor in one patient, the internal carotid artery was injured "inadvertently" (I do not like the word "inadvertently" which some of our assistants write in their operative reports: "the pleura was inadvertently opened" or "there was an inadvertent rupture of an artery"—I do not like it) and this patient had an ipsilateral hemiplegia because she had hypertension and had had a previous cerebral vascular accident on the opposite side. You can imagine what the result was in that particular case.

Then, the other patient had a very large tumor which extended from the middle fossa anterior to the pons and was wrapped around the basilar artery, and again "inadvertently" the basilar artery was opened with a similar result.

The third patient, a severe diabetic, was perfectly normal on the 4th postoperative day, but on the 5th day he died suddenly. Autopsy was performed with the report that the patient died from an unexplained cause. You can figure that one out. Of course, it is an operative death.

I do feel that it is important to record some of these rare tumors. Perhaps in the larger tumors that involve the cavernous sinus one should be inclined to leave just a bit of the tumor since it is slow growing.

I want to congratulate Dr. Svien on his paper.

CIRCULATORY DYNAMICS OF THE CANINE SPINAL CORD

TEMPORAL PHASES OF BLOOD FLOW MEASURED BY FLUORESCEIN AND SERIOROENTGENOGRAPHIC METHODS*

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IN THE course of studies of experimental angiography in dogs, it became evident that the spinal cord is especially vulnerable to injury by roentgen contrast agents, and that this injury is modified strikingly by the position of the animal during an injection.[13,16] It was readily apparent that the peculiar features of the blood supply and the caudad anatomical situation of the animal cord are important factors in the production of the injury. The postural effect, with the cord being protected when an animal is prone and rendered most vulnerable with the animal supine, could be explained in theory by differential gravitational flow of a medium heavier than blood.[7,13,16] An alternate possibility arises that the unusual susceptibility of the spinal cord to this injury, and the modifying effect of position could be explained by intrinsic features of the circulation of the spinal cord. For example, it is conceivable that the circulation time of the spinal cord is inherently slower than that of the brain, rendering the exposure time to the contrast substance critically long. Consistent with this hypothesis is the observation that the spinal cord is relatively resistant to circulatory stasis such as that produced by aortic occlusion.[1,11] Another possibility is that the modifying effect of posture is related to an alteration of speed of blood flow. With the animal supine, a slowing of the circulation of the cord by venous stasis or by arterial constriction is possible theoretically.[3] The importance of prolonged application time to the production of contrast medium injury of the brain has been emphasized repeatedly by Broman *et al.*[4,5] To resolve the problems raised above, information is needed concerning the circulation time of the spinal cord and, in particular, the time of exposure of the circulation and parenchyma of the spinal cord to an injected mass of contrast substance in its first circulation.

These few basic facts about spinal cord circulation are unknown. From a consideration of the anatomic arrangement of the blood supply of the

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cord, with its variable and numerous arterial and venous radicles, it is evident that the quantitative technics used in the measurement of cerebral blood flow are not applicable to this structure. However, the pattern of both the extrinsic and intrinsic vessels of the cord[1,6,9,13,15,16] renders it as readily approachable as the brain by technics designed to study temporal phases of the circulation, and perhaps even better adapted for the study of features of segmental or regional blood flow. The present report describes experiments designed to obtain this fundamental information, and the results of these studies.

EXPERIMENTAL

The principal method for the study of the vascular dynamics of the spinal cord consisted of a modification of the fluorescein technic used by Minard[14] to study the cerebral circulation time.

Adult dogs of varied breeds were used for these studies. With an animal under Nembutal anesthesia a laminectomy was performed, exposing from 3 to 5 cm. of lumbar cord. The dura mater was incised and reflected laterally, care being taken to avoid perforating the arachnoid membrane. In each animal the vascular network of the dorsal surface of the cord was carefully studied under a Zeiss technoscope at magnifications ranging from 10× to 60×. Animals demonstrating circulatory abnormalities such as the sludging phenomenon,[10] traumatic subarachnoid hemorrhage, or a loss of the subarachnoid space consequent to perforation were not used in critical observations. For further observations a magnification of 16× was generally used. A polyethylene catheter was inserted into the femoral artery and passed retrograde into the abdominal aorta until the tip reached to approximately the level of the umbilicus. For the indicator substance a 0.5 per cent Na fluorescein solution in physiological saline was used. This substance was injected manually in doses of 1 cc. per kg. at a rate of approximately 5 cc. per sec., this dose and injection time being based upon previously reported studies of contrast medium injuries.[13,16] The fluorescence of the dye was activated by exposure to a long wave (3660A°) ultraviolet lamp (Ultraviolet Products, Inc.) in a dark room. Care was taken during these tests to reduce the exposure to ultraviolet light to a minimum time and to keep the field of observation protected from light and moistened with physiological saline during interval periods. The circulatory events were timed by being directly recorded upon a Sanborn polyviso recorder through a remote control switch activated by the observer. During these studies continuous blood pressure tracings obtained from a catheter in the contralateral femoral artery were also recorded on this instrument. Animals exhibiting hypotension were eliminated from these studies of the normal circulatory dynamics. Parallel studies of the canine cerebral circulation were made through a 1.8 cm. trephine opening, under conditions similar to those of the spinal cord studies. For these observations a catheter was anchored in the external carotid artery in a retrograde direction, and the vessel was ligated distally. Na fluorescein in 0.5 per cent solution was again used as the indicator substance, with the test dose generally being 1 cc. per kg. administered at approximately 5 cc. per sec. In a few animals, doses of 0.2 to 0.4 cc. per kg. were also used.

The following variables of blood flow were recorded in each animal studied:

The appearance time—defined as the period elapsing between the beginning of the injection of the indicator dye and its appearance in the arterial circulation of the central nervous system.

The circulation time—defined as the period elapsing between the entry of the indicator agent into the pial arterial circulation and its appearance in the pial venous network at the site of observation.

The arterial exposure period—defined as the total time during which the indicator substance was visualized in the arterial circulation of the central nervous system.

The regional venous exposure period—defined as the total time during which the indicator material was visualized in the regional venous circulation of the central nervous system.

The regional application period—defined as the total time elapsing between the entry of the indicator dye into the arterial circulation of the central nervous system and its disappearance from the regional venous circulation of the central nervous system.

The readily defined arterial and venous components of the pial circulation served as reference points for these observations. In the spinal cord, particular care had to be exerted to exclude the dorsal medullispinal vein from these observations. This main dorsal venous channel, which carried the longitudinal in addition to the segmental venous drainage, remained fluorescent far longer (ca. 5 to 10 sec.) than did its tributary veins and therefore could not be used as an indicator of the regional venous exposure time. This vessel constituted such a prominent component of the vascular network that in most studies its period of fluorescence was also separately recorded. Thus, two more variables of circulation time were obtained, namely: the *general venous exposure period*, which encompassed the period of fluorescence of all pial veins; and the *general application period*, encompassing the period between the entry of the dye in the arterial circulation and its disappearance from the dorsal medullispinal vein. In the cerebral studies it was also necessary to separate the regional or local from the general venous drainage, but the latter component did not constitute so prominent a feature in these observations.

The following series of studies was made with the fluorescein indicator method:

1. Fifty observations of the spinal cord circulation in 10 dogs in the prone position.
2. Thirty observations in 5 dogs comparing the effect of the prone, lateral, and supine postures upon the circulatory dynamics of the spinal cord.
3. Twenty observations of the cerebral cortical circulation in 4 dogs.
4. Thirty-two observations in 4 dogs comparing the circulatory dynamics of the spinal cord to the brain.

As part of a study of broader scope a preliminary test was also made of the usefulness of roentgen contrast agents for the determination of the cir-

culation time of the spinal cord. For these studies 70 per cent Hypaque, 35 per cent Diodrast, and 25 per cent Thorotrast were used in 30 cc. doses, each agent being tested in one dog. These substances and the doses and concentration were chosen on the basis of previous tests showing that they yielded excellent angiographic contrast, yet exerted no toxic convulsant effect upon the cord. The events of the injection were recorded by serial roentgenograms made at 0.5-sec. intervals for a period of 13 sec. with a Schonander automatic film changer. This apparatus required that the dogs be positioned on the side during the injection. In the roentgenographic observations of spinal cord circulation the anterior spinal artery served as the arterial reference point. For the venous reference point an attempt was made to visualize the dorsal or ventral medullispinal veins of the cord and, failing in this, the radicular veins or the anterior internal vertebral venous plexus.

RESULTS

Fluorescein Studies. The fluorescence produced by the circulating dye was of brilliant quality and could be followed readily under microscopic observation. The advancing wave front of the fluorescein was sharply defined, both in the a rterial and venous circulations. The arterial fluorescence faded quickly, with a clearly perceptible end point. The decay of venous fluorescence was more gradual, but was still readily defined in the large veins constituting the regional drainage of the spinal cord. In the dorsal medullispinal vein the longer period of fluoresence faded so slowly that the end point was difficult to remark, making this reference point a less sensitive one than that of its tributary veins. Recirculation of the dye produced so feeble a fluorescence that it rarely interfered with these visual observations.

In these studies the directional pattern of blood flow over the stretch of the posterior surface of lumbar cord exposed for observation was particularly well demonstrated by the advancing wave front of fluorescein. The posterior arterial flow of the lumbar cord, as Bolton[2] has demonstrated in injection studies, was predominantly vertical, and, except for the loops where the arterial trunk divided into ascending and descending branches, the flow was craniad. The posterior venous circulation exhibited two directional features. The regional or segmental drainage was generally transversely oriented, although individual large veins contributing directly to the major posterior trunk presented a slanting craniad or caudad pattern. The directional flow through the longitudinally oriented dorsal medullispinal venous trunk was predominantly caudad.

The circulation time for the spinal cord ranged from 1.9 to 3.4 sec., compared to 0.9 to 2.0 sec. for the cerebral cortex. The regional application time for the spinal cord varied between 6.2 and 11.1 sec. for the spinal cord and 6.0 to 12.7 sec. for the cerebral cortex. Generally, it could be stated that the circulation time for the spinal cord was approximately twice as long as for the brain, but that the exposure time was approximately equal

TABLE 1

Comparison of temporal aspects of circulation through spinal cord and cerebral cortex of a dog (#13) measured with 0.5 per cent Na fluorescein indicator in 1 cc./kg. doses

Observation No.	Test Site	Circulation Times (Seconds)						
		Appearance Time	Circulation Time	Arterial Exposure Period	Regional Venous Exposure Period	Regional Application Period	General Venous Exposure Period	General Application Period
1	Spinal cord	3.5	2.8	6.1	6.8	9.6	12.5	15.3
2	Spinal cord	4.2	2.2	5.8	7.8	10.0	15.8	18.0
3	Spinal cord	3.7	2.5	5.0	6.9	9.4	18.0	20.5
4*	Brain	0.9	1.4	1.4	8.8	10.2	14.4	15.8
5	Brain	1.2	1.6	2.4	7.5	9.1	13.2	14.8
6	Brain	1.2	1.1	1.9	9.0	10.1	13.6	15.7
7	Brain	0.9	1.4	1.4	8.7	10.1	14.2	15.6
8	Brain	0.8	1.2	1.2	7.3	8.5	12.9	14.1

* Dose = 0.33 cc./kg.

in these two structures. It was also demonstrated that posture exerted no significant influence upon the measured temporal phases of blood flow through the spinal cord. Although the range of variation appeared rather broad in this series, it was generally observed that in a given animal the speed and duration of flow of the indicator agent were remarkably constant. For this reason individual experiments were considered to have more significance than the mass of data compiled. An illustrative experiment comparing the rate of blood flow through the spinal cord and the cerebral cortex is presented in Table 1, and in Table 2 an experiment testing the effect of posture upon the circulatory dynamics of the spinal cord is shown. These studies illustrate the degree of agreement and the range of variation encountered in studies with individual animals.

Roentgenographic Studies. The significant value derived from the seria

TABLE 2

Temporal aspects of blood flow in spinal cord of a dog (#11) in varied postures, measured with 0.5 per cent Na fluorescein indicator in 1 cc./kg. doses.

Observation No.	Position	Circulation Times (Seconds)						
		Appearance Time	Circulation Time	Arterial Exposure Period	Regional Venous Exposure Period	Regional Application Period	General Venous Exposure Period	General Application Period
1	Prone	3.1	2.0	4.4	4.7	6.7	15.3	17.3
2	Prone	2.7	2.0	4.8	8.1	11.1	15.3	17.7
3	Side	2.3	1.9	4.6	5.1	7.0	9.7	11.6
4	Side	2.3	1.9	4.8	5.1	7.0	12.9	14.8
5	Supine	3.5	2.3	4.8	5.7	8.0	10.3	12.6
6	Supine	3.4	2.2	4.4	5.6	7.8	11.1	13.3

angiographic studies was the indirect figure of 4 sec. for the circulation time of the spinal cord. The contrast agent appeared in the anterior spinal artery immediately after the end of the injection (Figs. 1 and 2). This trunk remained visible for 3.5 sec. but the radiopaque medium could not be demonstrated in the posterior arterial trunk or more peripherally in the anterior arterial tree or in the intrinsic or pial venous circulation of the spinal cord. The anterior internal vertebral venous plexus began to fill 4 sec. after the anterior spinal artery (Fig. 3) and showed a gradually deepening opacity over the next 3 sec. (Fig. 4). Recirculation of the radiopaque media, particularly the Thorotrast, obscured the terminal features of the first circulation of these agents.

DISCUSSION

These observations confirm the findings of Minard concerning the value of fluorescein as an indicator agent in the study of the vascular dynamics of the central nervous system. The high degree of fluorescence obtained by low dilutions allows repetitive studies to be made at close intervals without the production of toxic effects. The barrier mechanisms between blood-brain and blood-cerebrospinal fluid compartments render the brain unique among the organs of the body in that progressive development of background parenchymal fluorescence, which prevents repeated tests upon somatic structures, is negligible over long periods of observation.

While it is recognized that the anesthetic state lowers blood flow in the central nervous system,[12] and paralyzes the mechanisms regulating central vasomotor tone,[8] it is considered that these observations upon the anesthetized dog allow an approach to an understanding of the circulatory dynamics of the spinal cord in a normal state. Under these experimental conditions the circulation time of 1.9 to 3.4 sec. and the regional exposure time of 6.2 to 11.1 sec. obtained for the spinal cord indicate that no striking difference in metabolic activity exists between the cord and the brain, for which a circulation time of 0.9 to 2.0 sec. and a regional exposure time of 6.0 to 12.7 sec. were obtained. This cerebral circulation time is in remarkably close agreement with the 1.2–1.8 sec. figures obtained for the monkey by Minard,[14] which confirms the validity of these observations. Further confirmation is provided by the studies of Landau *et al.*[12] Using the radioactive biologically inert gas CF_3I^{131} this group measured the rate of blood flow per gram of tissue in specific structures in the nervous system of the cat. The rate of flow through the spinal cord gray matter was 0.63 cc./gm./min., compared to 0.88 for the association cortex and 1.25–1.38 for primary cortex. Comparative values for white matter were 0.14 for the spinal cord against 0.23 for the cerebrum. It would appear, then, that, using these figures as indicators of physiological activity, the spinal cord maintains a metabolic rate approximately one-half to two-thirds the level of the cerebrum. On this basis it is difficult to understand the reason for the experimentally demonstrated resistance of the spinal cord to circulatory stasis. The most likely explanation

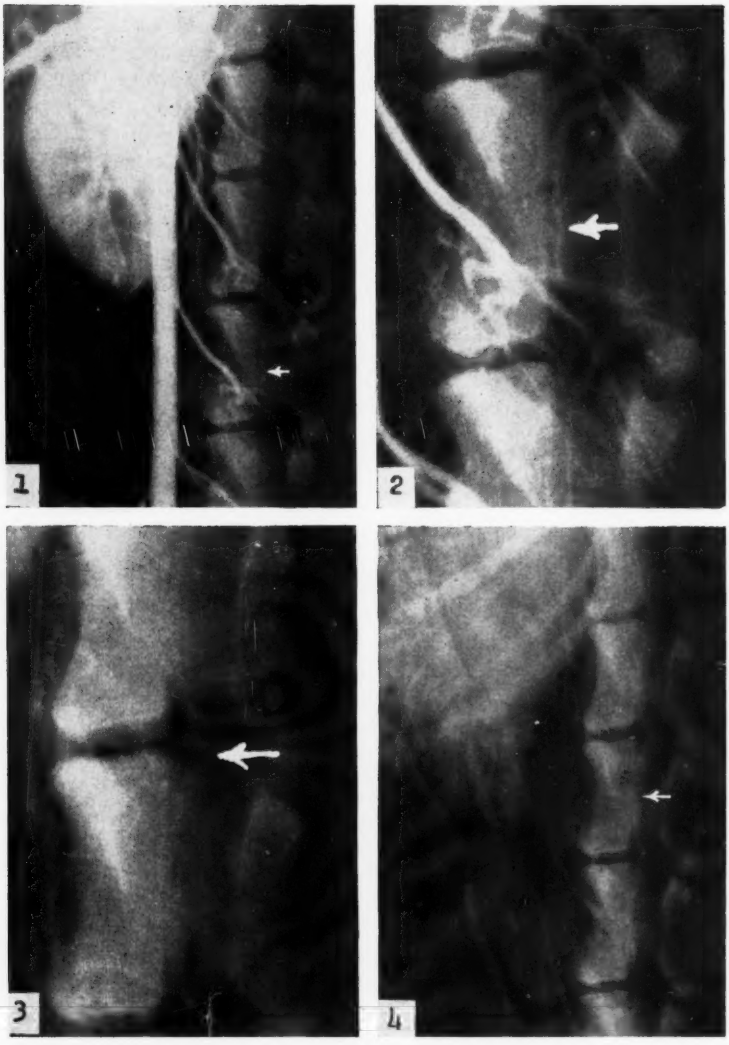

FIG. 1. Aortogram, early phase, showing first appearance of Diodrast in anterior spinal artery (arrow). This picture was made at zero time with reference to Figs. 3 and 4.

FIG. 2. Enlargement of Fig. 1 showing the filling of the anterior spinal artery.

FIG. 3. An enlarged field showing the disappearance of contrast medium from the anterior spinal artery and the early visualization of the anterior internal vertebral vein at 4 sec.

FIG. 4. Maximal opacification of the anterior interior vertebral vein is observed at 7 sec.

for this phenomenon resides in the technical difficulty in producing absolute ischemia of the spinal cord.[1,11]

From the recent review of Adams and van Geertruyden[1] of the anatomy of the vascular system of the spinal cord, drawn heavily from the classical study of Kadyi,[9] and from our own observations,[11,15] it is clear that the desegmented vascular pattern of the canine cord renders it directly comparable to that of man. From these studies it is clear that the canine spinal cord has no intrinsic features that render it particularly susceptible to the effects of contrast agents. It is even possible that the slightly slower circulation time is explicable on the basis that the observations, made upon the vascular tree of the posterior aspect of the cord, which largely represents the flow through white matter,[15] reflect only the slowest aspects of the cord circulation. Further, in the supine position, which renders the dog most vulnerable to the effects of contrast agents, no alteration of the rate of circulation or of the application time of a standard injection has been observed. Thus, it is evident that the factor of prolonged duration of exposure of the vascular bed of the spinal cord plays no part in the production of increased vulnerability of the spinal cord to contrast medium injury. In the dog the caudad anatomical position of the cord[16] and the differential gravitational flow of radiopaque agents having a higher specific gravity than blood[7,16] remain as the only factors of significance with regard to the augmentation of this injury.

In addition to establishing the value of a modified fluorescein technic for the demonstration of the normal circulatory dynamics of the cord, these studies also provide fundamental data prerequisite as a background for the study of blood flow through the cord during the passage of nontoxic and toxic concentrations of contrast agents. Finally, they serve to encourage the exploration of instrumentational methods that will eliminate from these studies the factor of human error, a need that has been clearly demonstrated by Minard. Both of these latter projects are now under way in our laboratories.

While the anterior spinal artery and the anterior internal vertebral venous plexus can be visualized by the serial radiographic technic, the failure to demonstrate the pial venous network of the spinal cord eliminates this method as a means of directly recording the circulation time of the cord. Considering that a longer circulatory pathway is measured when the anterior internal venous plexus is used as the venous reference point the observed circulation time of 4 sec. is consistent with the results of the direct fluorescein studies.

SUMMARY

The temporal phases of blood flow through the canine spinal cord and cerebral cortex have been measured by a fluorescein indicator method. The circulation time of the spinal cord varied from 1.9 to 3.4 sec. and that of the brain 0.9 to 2.0 sec. The duration of exposure (regional application time) to a standard dose of indicator dye was 6.2 to 11.1 sec. for the cord and 6.0 to

12.7 sec. for the cerebral cortex. The influence of posture upon the measured variables of the circulation has been tested, and found to be negative. The significance of these observations upon the interpretation of the mechanisms of contrast medium injury to the nervous system has been discussed. It is concluded that the cord has no intrinsic features of its circulation rendering it particularly susceptible to contrast medium injury.

An attempt was also made to measure the rate of blood flow through the spinal cord by serial roentgenograms. While visualization of the anterior spinal artery was possible, the medullispinal veins could not be demonstrated. Hence, this technic was not adequate as a direct method of measurement of the rate of blood flow in the spinal cord.

REFERENCES

1. Adams, H. D., and van Geertruyden, H. H. Neurologic complications of aortic surgery. *Ann. Surg.*, 1956, *144:* 574–609.
2. Bolton, B. The blood supply of the human spinal cord. *J. Neurol. Neurosurg. Psychiat.*, 1939, n.s. *2:* 137–148.
3. Brecher, G. A. Venous return. *New York: Grune & Stratton*, 1956, 148 pp.
4. Broman, T., Forssman, B., and Olsson, O. Further experimental investigations of injuries from contrast media in cerebral angiography. Summation of various injurious factors. *Acta radiol., Stockh.*, 1950, *34:* 135–143.
5. Broman, T., and Olsson, O. Experimental study of contrast media for cerebral angiography with reference to possible injurious effects on the cerebral blood vessels. *Acta radiol., Stockh.*, 1949, *31:* 321–334.
6. Herren, R. Y., and Alexander, L. Sulcal and intrinsic blood vessels of human spinal cord. *Arch. Neurol. Psychiat., Chicago*, 1939, *41:* 678–687.
7. Hol, R., and Skjerven, O. Spinal cord damage in abdominal aortography. *Acta radiol., Stockh.*, 1954, *42:* 276–284.
8. Ingvar, D. H., and Söderberg, U. A new method for measuring cerebral blood flow in relation to the electroencephalogram. *Electroenceph. clin. Neurophysiol.*, 1956, *8:* 403–412.
9. Kadyi, H. Ueber die Blutgefässe des menschlichen Rückenmarkes. Nach einer im xv. Bande der Denkschriften der math.-natur. Classe der Akademie der Wissenschaften in Krakau erschienenen Monographie, aus dem Polnischen überzetzt vom Verfasser. *Lemberg: Gubrynowicz & Schmidt*, 1889, 152 pp.
10. Knisely, M. H. An annotated bibliography on sludged blood. *Post Grad. Med.*, 1951, *10:* 15–24; 80–93.
11. Krogh, E. Studies on the blood supply to certain regions in the lumbar part of the spinal cord. *Acta physiol. scand.*, 1945, *10:* 271–281.
12. Landau, W. M., Freygang, W. H., Jr., Roland, L. P., Sokoloff, L., and Kety, S. S. The local circulation of the living brain; values in the unanesthetized and anesthetized cat. *Trans. Amer. neurol. Ass.*, 1955, 125–127.
13. Margolis, G., Tarazi, A. K., and Grimson, K. S. Contrast medium injury to the spinal cord produced by aortography. Pathologic anatomy of the experimental lesion. *J. Neurosurg.*, 1956, *13:* 349–365.
14. Minard, D. Fluorescein studies of circulation time in monkeys with the lucite calvarium. *Naval Medical Research Institute, Bethesda, Md.*, Project N.M. 007 081.07.04, 1950, 1–14.
15. Suh, T. H., and Alexander, L. Vascular system of the human spinal cord. *Arch. Neurol. Psychiat., Chicago*, 1939, *41:* 659–677.
16. Tarazi, A. K., Margolis, G., and Grimson, K. S. Spinal cord lesions produced by aortography in dogs. *Arch. Surg., Chicago*, 1956, *72:* 38–47.

GLIOMAS OF THE OPTIC PATHWAYS IN CHILDHOOD

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A SMALL series of cases of optic pathway gliomas is being reported for two reasons: first, because their proper treatment is still a controversial subject and second, because of the importance of early recognition in permitting possible surgical cure instead of palliation. Gliomas of the optic pathways make up a relatively small portion of neoplasms of the brain. Martin and Cushing[27] reported an incidence of 0.84 per cent in a series of 826 intracranial tumors. Taveras *et al.*[33] found 34 cases in a series of 2000 gliomas (1.7 per cent). If the various reported series of optic pathway gliomas are inspected, it is found that approximately 75 per cent of these lesions occur in children under the age of 12.[5,7,13,17,20,31,32] In our clinic 13 cases of primary tumor of the optic pathways have been encountered among a series of 256 intracranial tumors in young children during the past 10 years. This is an incidence of 5.1 per cent in the total series and 7.0 per cent if only the gliomas are considered.

Much emphasis has been placed on the fact that these tumors have only a slight malignant potentiality. Hudson[17] first pointed out that even after incomplete removal, patients may survive for 20 years or more without evidence of progression of the tumor. This has been confirmed by the experience of others.[25] McFarland and Eisenbeiss[23] have even stated that if such a tumor is only partially removed it probably will not continue to grow. As recently as 1954, Marshall[26] said, "On theoretical grounds complete excision might be desirable, but recurrence after excision of the major portion is very uncommon." Many of these statements have been made by ophthalmologists on the basis of an anterior exploration of the orbit, an approach that makes it difficult to visualize the limits of the tumor. Also, since it has been well established that many of these gliomas grow very slowly, the occurrence of intracranial symptoms 10–15 years after the original operation might not come to the attention of the original surgeon. Posner and Horrax[30] have pointed out that even after known intracranial extension, these patients may survive for a long time, their only symptom being loss of vision.

Analysis of the 13 patients seen at the Children's Medical Center in the past 10 years with a verified diagnosis of optic pathway glioma reveals that on the basis of symptomatology and physical findings they fall into two groups, depending on whether the tumor is predominantly intraorbital or intracranial. This differentiation was first proposed by Dandy,[8] but has not been used by many subsequent authors. The following case histories illustrate some of the important differences between these two groups.

CASE REPORTS

Intraorbital Glioma

Case 1. M.C., a 4½-year-old girl, was admitted to the Children's Medical Center because of increasing prominence of the left eye of 2 weeks' duration (Fig. 1). She had had no headaches and had noted no change in her visual acuity. Although there was some increased resistance to palpation of the globe, no tumor mass was palpable. The movement of the eye was unimpaired. Early papilledema was present in the left eye; the right fundus appeared normal. Visual acuity was diminished to 20/70 in the left eye and was normal in the right. Slight peripheral constriction was found in the visual field of the left eye, but the patient could not cooperate for detailed examination. There were no other abnormal findings and no evidence of von Recklinghausen's disease.

FIG. 1. *Case 1.* Appearance on admission to the hospital. Note the slight prominence of the left eye which was the only reason for seeking medical attention.

Roentgenograms showed the left orbit to be slightly larger than the right, but the optic foramen was normal. Films of the skull were otherwise negative. A left carotid arteriogram showed no evidence of displacement of the intracranial vessels and no abnormality of the orbital vascular pattern.

A left transfrontal craniotomy was performed. On exploring the orbit, a diffuse reddish swelling of the optic nerve was found. Further exploration demonstrated that this tumor lay entirely within the orbit. The nerve was first amputated from the globe. Intradural exploration of the chiasm was then performed and failed to show any abnormality of the intracranial portion of the optic nerve. The nerve was divided just in front of the chiasm, pulled through into the orbit, and removed. Since subsequent pathological examination showed that the tumor extended to the globe, the eye was enucleated 16 days later and the patient was fitted with a prosthesis.

Histologically, the resection extended 1 cm. behind the most proximal edge of the tumor and thus the neoplasm was considered to have been completely removed. The microscopic picture of this lesion was characteristic of an optic pathway glioma (Fig. 2). There was a matrix of fine neuroglial fibrils in interspaces of a delicate reticulated syncitium. The tumor cells were small with oval or elongated nuclei, some of which were darkly stained. Many of the cells showed vacuolization of the cytoplasm.

It is now 8 months since operation and the patient is asymptomatic (Fig. 3).

Intracranial Glioma

Case 2. A.P., a 7-year-old child, had a history of a squint of her left eye which

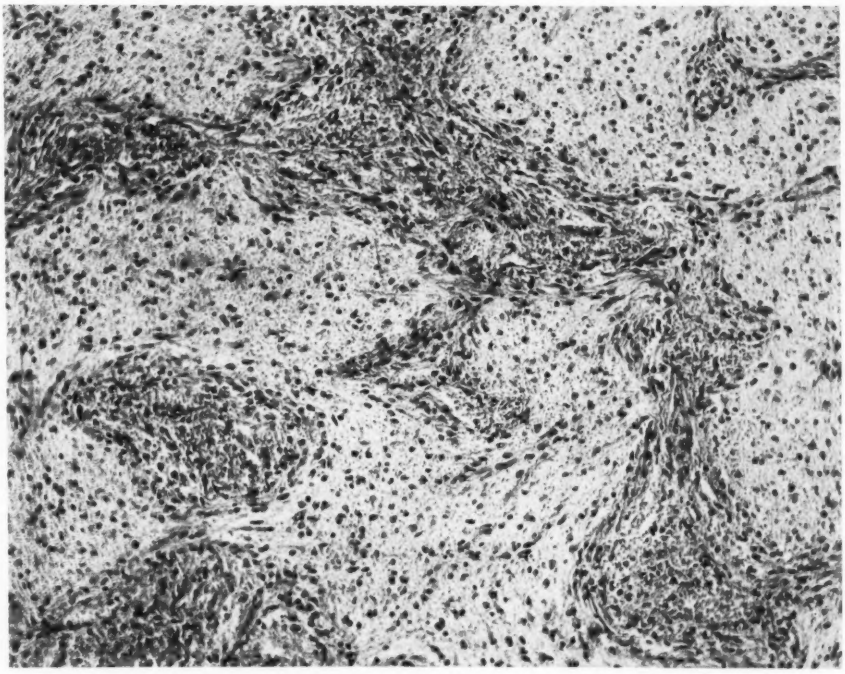

FIG. 2. *Case 1.* Histologic appearance of optic nerve glioma. Many of the bundles of nerve fibers are preserved and the tumor cells show no malignant tendency. There is marked hypertrophy of the pial septa. Toluidine blue-eosin stain, ×125.

had first been noted 3½ years previously. Shortly after the onset of this symptom language difficulty developed and within a period of a few weeks speech became incomprehensible. She was examined by a doctor at that time who found that she could not see with her left eye. These findings did not progress and no specific therapy was instituted. Two weeks before admission to the Children's Medical Center she began to have headaches for the first time which were associated with early morning vomiting. On physical examination she had weakness of her left lateral rectus muscle, but there was no proptosis of either eye. Papilledema was present in the right eye. The left fundus showed marked optic atrophy, and the eye was completely blind. Scattered over her body were café-au-lait spots and small subcutaneous masses characteristic of von Recklinghausen's disease (Fig. 4).

Roentgenograms showed the left optic foramen to be twice as large as the right. There was some erosion of the apex of the petrous ridge on the left as well as some destruction of the inferior surface of the anterior clinoid process.

Operation disclosed a large tumor in the middle fossa arising from the left optic nerve. This was adherent to the left internal carotid artery and to the right optic nerve, but could be dissected free from these structures leaving them intact. The tumor extended into the orbit, but faded out posteriorly in the chiasm. The chiasm was amputated flush with the right optic nerve in order to spare the temporal retinal fibers on this side. The intracranial portion of the tumor was thus grossly removed.

FIG. 3. *Case 1.* Eight months following operation. The patient is asymptomatic and has a prosthesis in place of the left eye.

FIG. 4. *Case 2.* In addition to her ocular findings this patient showed the café-au-lait spots of von Recklinghausen's disease.

Fourteen days later the left orbit was explored through the previously made frontal craniotomy and the tumor was found to extend into the globe itself. The intraorbital portion of the optic nerve and the tumor were then removed. Six months later the globe was enucleated.

Pathologically this tumor was designated as a glioma of the optic pathway. The lesion had been completely removed as far as could be determined by gross examination, but there is a possibility of some remaining microscopic involvement in the left optic tract posterior to the chiasm.

The child did well following surgery. The right nasal field of vision is still intact. One and a half years following her operation she began to show secondary sexual characteristics (age 9 years). It is now 3 years since the operation and $6\frac{1}{2}$ years since her symptoms first developed—she has occasional headaches, but is otherwise asymptomatic.

SYMPTOMATOLOGY

The average age of our patients is $4\frac{1}{3}$ years; there is an equal distribution of sex. The young average age is ascribable partially to the fact that these

patients came entirely from a pediatric population; however, this has also been the experience of other authors who have drawn their patients from a more widely scattered range of age.

The presenting symptoms depend on the portion of the optic pathway that is first involved, and although the same pathological process may be seen either intraorbitally or intracranially, the clinical picture varies depending on the origin of the tumor. Within the primarily intraorbital and primarily intracranial groups there is a certain uniformity of findings.

In the intraorbital group there are 4 children. None of these had evidence of von Recklinghausen's disease. The initial symptom in all patients was proptosis of the affected eye. This proptosis, as noted by other authors, is nonpulsatile and is usually an axial protrusion of the globe directly forward. If displacement does occur as the tumor enlarges it is most frequently inferior and lateral. Christensen and Andersen[5] have said that there is usually no impairment of extraocular movements. Although this would seem to be a reasonable statement, 2 of our 4 patients had some limitation of ocular motility. This was not marked in relation to the degree of proptosis, however. In general, among orbital lesions, optic nerve gliomas are certainly much less apt to produce early alteration in eye movements than lesions of the bony orbit or of the extraocular muscles themselves.

These patients were seen early—the time after initial symptoms being 2, 4, and 5 weeks and 8 months. All had papilledema in the affected eye. Other writers have reported optic atrophy, but it may be that our patients were seen at such an early stage that this had not had sufficient time to develop. Two of these patients had decreased visual acuity on physical examination although they were unaware of it prior to admission to the hospital. Visual field examinations are unsatisfactory in this age group, but as far as could be determined none of the children had any obvious field defect. Although it is known that diffusely infiltrative tumors of this type often produce little interference with neurologic function, it may be that in older patients minor alterations in the visual fields can be found.

Palpation of the orbit is abnormal in the presence of an orbital glioma. Usually the tumor cannot be felt, but examination will reveal increased resistance of the globe to posterior displacement. Although papilledema was present in all, none of the patients in the intraorbital group showed other evidence of increased intracranial pressure. The papilledema under these circumstances is caused by intraorbital rather than intracranial obstruction of venous drainage from the retina.

Among the 9 patients in the intracranial group the symptoms and signs were quite different. In general, the history was somewhat longer than in the purely intraorbital group, averaging 20 months. Six patients had decreased visual acuity as a presenting complaint. Again the determination of visual field defects was extremely difficult because of the young age of the patients. The finding of irregular bitemporal field defects as evidence of involvement of the intracranial portion of the nerve and chiasm has been

well described by Cushing[7] and it is probable that if reliable examination could have been done this would have been evident. Only 2 patients presented with proptosis, and both of these had intraorbital as well as intracranial tumor. Five of them had optic atrophy in the affected eye or bilaterally, 1 had bilateral papilledema, and 2 had atrophy in one eye and papilledema in the other.

Increased intracranial pressure was a frequent sign if the tumor was located intracranially. In addition to the eye signs, 3 of these children had a positive Macewen's sign and 3 had an unsteady gait which was probably related to increased pressure. Further evidence of intracranial tumor are signs of hypothalamic dysfunction and occasionally other symptoms caused by involvement of structures in the neighborhood of the optic chiasm. In 3 of our patients sexual precocity developed—at ages 7, 8 and 9.

Four of the children in this group have von Recklinghausen's disease. This corresponds well with the findings of other authors who have reported that about one-third of patients with optic pathway gliomas showed stigmata of generalized neurofibromatosis.[10,20] One of these patients had a sibling with von Recklinghausen's disease and an optic nerve glioma.

RADIOGRAPHIC FINDINGS

It should be emphasized at the outset that gliomas of the optic pathways may be present either intracranially or intraorbitally without any alteration in the appearance of the plain roentgenogram. It should likewise be stated that the radiographic signs produced by these tumors are not pathognomonic: they may be duplicated by other lesions in this area.[22,28]

Plain orbital roentgenograms in patients with intraorbital gliomas may show evidence of enlargement of the optic foramina. The average normal foramen is bean-shaped with dimensions of 4.1 by 4.65 mm. It is always necessary to compare both sides in order to discover minor variations. It must be remembered, however, that the foramina are absolutely symmetrical in only 45 per cent of the normal population, in 40 per cent they may differ as much as 10 per cent in size, and in the remainder variations up to 20 per cent may be noted.[14] The contour is normally smooth; therefore, any irregularity, hyperostosis, or erosion is apt to be pathological. The commonest abnormal finding in patients with gliomas of the optic nerve is a smooth, circular expansion of the optic foramen (Fig. 5). It is, of course, necessary to have films of good technical quality in order that the contour and dimensions of the two foramina can be compared. Enlargement of the optic foramen also is found occasionally with other lesions, and therefore, although it is most frequently seen with optic nerve tumors, it is not always pathognomonic.[1]

Various ancillary aids to roentgenographic diagnosis have been suggested. Orbital tomography has been said to give additional information about small intraorbital masses.[29] This has been modified by injecting air into the orbit prior to tomography.[11] Such a technique may delineate small areas of

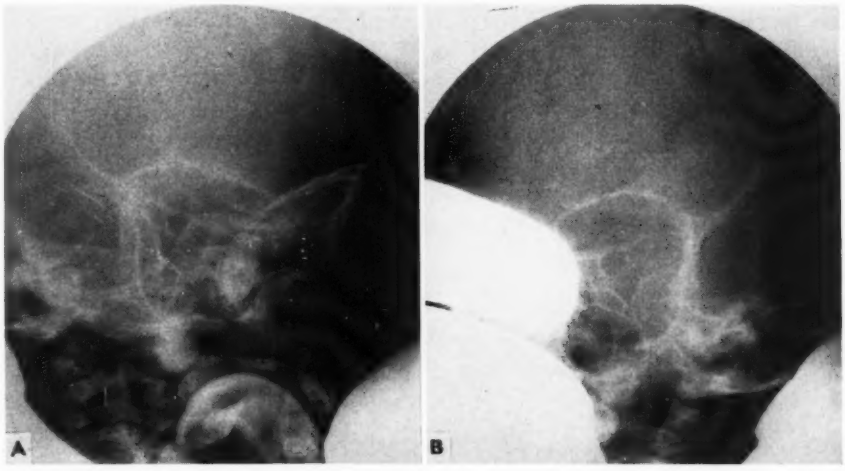

FIG. 5. Roentgenograms of the optic foramina in a patient with an intraorbital glioma. (A) Normal right foramen measures 4.0 mm.×4.5 mm. (B) Expanded left foramen measures 7.5 mm.×8.0 mm. Note the more circular shape of the foramen.

enlargement of the optic nerve which will allow differentiation between gliomas and other intraorbital tumors. In the use of tomographic techniques in this region the possible hazards of excessive roentgen exposure should be evaluated carefully, especially in young people. In our clinic we have had no experience with this procedure and we doubt the wisdom of its use as a routine diagnostic maneuver. It has been suggested that carotid angiography may be of assistance in the diagnosis of orbital tumors.[16] This is primarily of value in differentiating optic nerve tumors from the more vascular orbital lesions, particularly angiomata and metastatic neoplasms.

In 2 patients in our intraorbital group there was enlargement of the optic foramen. The tumor in these cases did not extend all the way through the optic canal (Fig. 6). If this radiographic sign had been used as a contraindication to surgery, as has been suggested,[33] the opportunity for curing these children would have been lost. Another of the intraorbital tumors caused slight enlargement of the entire orbit, the foramen being normal. In 1 patient the plain roentgenograms were entirely normal. Angiography was performed in 2 of the patients with intraorbital tumors and in both it was normal.

FIG. 6. Gross specimen of the tumor removed from the orbit of the patient whose roentgenograms are shown in Fig. 5. This tumor enlarged the optic foramen without extending intracranially.

In the intracranial group the plain roentgenograms of the skull proved to be abnormal in all cases. In 4 of these patients the optic foramen was enlarged and in 2 the sella turcica was eroded. The characteristic deformity of the sella turcica associated with glioma of the optic pathway was first described by Martin and Cushing in 1923.[27] This is a pear-shaped configuration of the bony contour produced by erosion of the tuberculum sellae and the anterior clinoid process (Fig. 7). In 3 patients both the sella turcica

FIG. 7. Characteristic deformity of the base of the skull produced by an intracranial glioma of the optic nerve eroding the tuberculum sellae.

and the optic foramen were altered. Therefore, in this group, all patients showed some abnormality in the plain films.

A further aid in defining the extent of these tumors is pneumographic examination of the basilar cisterns.[3,9] Such a technique may outline even a small mass lying in the region of the optic chiasm. Angiography also may be of some help, although deformities of the anterior portion of the circle of Willis would not be noted unless a rather large mass were present. If the patient has signs and symptoms of increased intracranial pressure it is likely that there is a mass obstructing the ventricular system; in these circumstances, ventriculography is the safest and most instructive manner in which to visualize the extent of the tumor.

PATHOLOGY

The optic nerve is not a cranial nerve in the usual sense, but, rather, an

extension of a tract of the brain and, therefore, it contains the same glial elements that are to be found elsewhere in the central nervous system. It might thus be expected that tumors arising in this region could derive from any of the glial cells of the brain, and this is probably the case. There have been convincing reports of glioblastoma multiforme originating within the optic nerve,[31] but as a rule gliomas of the optic pathways are more benign. Occasionally verified gliomas have been reported to involve the retina itself.[24,35]

Some authorities have considered that the majority of these tumors are derived from cells of the astrocytic series.[2,15] A great deal of evidence has been accumulated on the other hand through the use of special stains to show that the predominant cell is an oligodendrocyte rather than a spongioblast or astrocyte.[5,18] The possible oligodendrocytic origin of these lesions is of particular interest in view of the association of these tumors with von Recklinghausen's disease, as oligodendrocytes are probably the counterpart in the central nervous system of the Schwann cells in the peripheral nerve. Regardless of what the primary cell may be in this neoplastic process, the tumors seem to have a consistent histologic pattern and therefore they have been designated only as gliomas of the optic pathways rather than by a more specific histologic classification.

The usual pathologic picture seen in these lesions is generalized swelling of the nerve with hypertrophy of the pial septa. There is scant vascularization of the tumor and the walls of the vessels are usually normal. Within these tumors Christensen and Andersen[5] described four distinct cell types. There are small cells with round nuclei and delicate fibrils which seem to be identical with the normal oligodendroglia of the brain. As transition from these there are coarsely reticulated cells that are polygonal in shape with large dark nuclei and fewer fibrils. These cells are oligodendrocytes and are found typically in this type of neoplasm. There are also sickle-shaped cells of astrocytic origin and occasional large cells resembling neurones which sometimes have more than one nucleus. There are frequently some degenerative changes such as vacuolization of the cytoplasm.[34] In general, the histologic picture is one of a mature and benign glioma. This is verified clinically by their slow rate of growth and by the fact that they have shown no tendency to metastasize by way of either the blood stream or subarachnoid pathways.

All the tumors in this series conform reasonably well to the above description. There was no apparent difference between the intraorbital and intracranial tumors histologically. Likewise, the tumors in patients with von Recklinghausen's disease did not vary to any significant extent from the others.

As has been mentioned, in addition to this characteristic type of lesion there may occur typical tumors of the astrocytic series; more malignant neoplasms, such as glioblastomas, have also been reported. There were no such lesions found in our patients.

TREATMENT

These tumors were originally treated usually by ophthalmologic surgeons who exposed them anteriorly through the orbit. This was considered to be the safest method, which it probably was, 40–50 years ago, despite the fact that it was associated with a 10 per cent incidence of meningitis.[17] Although the danger of infection is less of a problem at present, there are certain major disadvantages of the anterior approach which pertain even today. By this route is not possible to expose the region of the optic foramen adequately, and extension of a tumor through the foramen cannot be seen—much less treated properly. The use of roentgen examination to exclude intracranial extension of a predominantly intraorbital lesion is not justified.[4] Therefore, it is not possible to be sure prior to operation whether the neoplasm is completely intraorbital. It does not seem reasonable to be satisfied with incomplete removal on the assumption that the remainder of the tumor can be expected to stop growing or regress spontaneously.

The statement has been made by Marshall[26] that operations in the region of the chiasm are not only dangerous, but often fatal because of damage to vital centers. The recent paper by Love and Benedict[21] in which they reported only 1 death in 35 patients who had removal of intraorbital tumors by the transcranial route would seem to refute this argument. The use of cortisone and adrenocorticotrophic hormone as an adjunct to the surgery of parasellar lesions has improved the morbidity and mortality of such procedures.[19] Certainly intracranial exploration of the chiasm for evaluation of extent of the tumor and the possibility of radical resection should nowadays carry a negligible risk.

With the exception of 2 patients who had had surgical treatment prior to coming under our care, a uniform operative approach has been used. With a presumptive diagnosis of glioma of the optic pathways a transfrontal exposure of the roof of the orbit and the region of the chiasm is performed. If there are definite clinical signs of intraorbital involvement, the roof of the orbit is removed extradurally. The orbit can thus be decompressed and explored adequately. If tumor is found in the nerve a resection up to the globe is done. Whether or not there is clinical evidence of intracranial extension it is wise to open the dura mater and visualize the nerve and chiasm. In most instances it is best to resect the optic nerve back to the chiasm. This does not give the patient any additional disability to that already sustained in the intraorbital procedure and does provide further assurance that the tumor has been completely removed. Indeed, on occasion, it may be possible to remove an involved portion of the chiasm itself if it is felt that this will insure complete excision of the neoplasm. Such a procedure may further impair the patient's vision in the opposite eye, but a hemianopic field of vision is not an intolerable neurologic defect, and is justified if complete removal can be accomplished. If, however, there is gross involvement of the entire chiasm or the optic tract so that complete removal is precluded, it is better to be more conservative and remove only sufficient tissue for biopsy. If, in

such a case, there is any remaining vision in the affected eye it is our feeling that careful decompression of the orbit and optic canal may give the best chance for improvement, or at least for delay in further visual loss. It is likely in this circumstance that the patient will have a reasonably long period of survival with slow progression of symptoms. There is certainly no justification for increasing disability by an operation that will not be curative.

It is only by this type of exploration that the tumor can be evaluated properly and those patients selected in whom complete surgical extirpation is warranted. Certainly at the present time surgery is the only definitive method of curing these patients. When complete removal of the intraorbital and intracranial portions of the tumor has been accomplished and it is found that the tumor has extended into the globe, enucleation followed by insertion of a prosthesis may be done by the ophthalmologist at a later date. It is possible to leave the eye in place if there is no neoplasm in the distal end of the nerve; however, in all of our patients enucleation proved necessary in order to achieve complete excision of the tumor.

In the 4 patients with purely intraorbital tumors the above method of therapy was used, and in all it was found histologically that the tumor had been completely resected. These patients are now living and well 1 month, 8 months, 5 years, and 8 years following operation. All of these patients have normal vision in the remaining eye.

Of the 9 patients in the intracranial group 2 died postoperatively. These were the 2 earliest cases in our series. In both there was found to be a large intracranial mass from which a biopsy was taken, no attempt being made to remove the entire tumor. Both of these children died within 3 days of their initial operation in status epilepticus. There was no evidence of operative complication found at postmortem examination and no adequate anatomical explanation was found for their death other than the large tumor extending posteriorly in the middle fossa together with a minimal degree of cerebral edema. These patients were both operated upon prior to the availability of cortisone.

The other 7 patients of the intracranial group are living, 6 of them with useful vision. The patient who is blind had already lost all vision prior to operation. One of these patients is 7 years from the time of his initial operation, 3 are 4 years, 1 is 2 years, 1 is 6 months, and 1 is 1 month. Unfortunately, most of these patients are too young to allow accurate objective measurement of visual acuity, but at least they are all carrying on without special visual or sight-saving aids. Three patients have subsequently shown sexual precocity, that is, adolescent secondary sexual characteristics developed before the age of 8 or 9. The others are either still too young or have shown no evidence of premature sexual maturation. No objective analyses of endocrine function have been carried out on the whole group.

Four of the 7 remaining patients had partial removal of the intracranial mass; 1 had a grossly complete removal of the intracranial mass as well as

removal of the intraorbital portion of the optic nerve and globe. Two patients had only exploration of the chiasm with no attempt to excise the lesion because it was found that there was extensive involvement of both optic nerves and chiasm. In those patients who have an inoperable intracranial mass causing increased intracranial pressure by blocking the anterior portion of the third ventricle, a cerebrospinal fluid shunting procedure may give symptomatic relief. This was done in 1 of our patients by performing a bilateral ventriculocisternostomy.

The use of roentgen therapy in these lesions is a subject of some debate. Evaluation of any such therapy is difficult because many of these tumors grow very slowly even without any definitive treatment. Thus, length of survival cannot in itself be used as a criterion of the efficacy of roentgen therapy. Many of the recorded statements must be regarded as clinical impressions, since many of the data on which they are based do not bear statistical scrutiny.

Duke-Elder[12] is of the opinion that roentgen therapy is of little value. Cohen[6] recognizes the fact that these tumors are very slowly growing, but in some cases he feels that radiation treatment seems to be of limited value. More recently Taveras *et al.*[33] reported that roentgen therapy is effective in the treatment of these lesions. However, of 17 patients with verified gliomas with adequate follow-up in their series only 4 were objectively improved (using the criterion of visual acuity). In the other 13 patients there was either no change or they continued to have slow progressive visual loss, as is usually expected. The most significant results were noted in a group of 9 patients in whom the diagnosis was made only on clinical and radiographic evidence. Of these, 6 patients were definitely improved clinically, and sometimes to a remarkable degree. It is felt, however, that such observations must be considered with reservation because of the occurrence of other lesions simulating these gliomas both clinically and radiologically, and thus possibly giving rise to misleading conclusions. Although this report indicates that roentgen treatment has been of value in certain cases, there is no proof of an appreciable effect on the majority of these lesions.

None of our patients with purely intraorbital lesions was given postoperative roentgen therapy since it was felt that total surgical excision had been accomplished. Among the intracranial group 1 was given a depth dose of 4850 roentgens to the region of the chiasm and involved orbit and 1 was given 2830 roentgens to the intracranial portion of the optic nerves and chiasm. There was no evidence of improvement of vision in either of these 2 patients; however, their signs have not progressed significantly 6 months and 4 years after treatment. In the 5 patients who had no therapy other than partial surgical excision the results are comparable. It would seem reasonable in our opinion, therefore, that if a glioma of the optic pathways cannot be completely excised it would be well to give roentgen therapy to the areas involved in the hope that it might be of some value in delaying further growth. Based on our own limited experience it cannot be assured that any improvement in neurologic status will take place after such treatment.

SUMMARY

Gliomas of the optic pathways are slow-growing, non-metastasizing brain tumors occurring predominantly in childhood. The presenting signs depend on whether the neoplasm originates from the intraorbital or intracranial portion of the optic pathway.

Progressive unilateral proptosis combined with decreasing visual acuity is the common presenting clinical picture in the primarily intraorbital gliomas. Increased intracranial pressure combined with decreasing visual acuity is the characteristic feature of primarily intracranial optic pathway gliomas.

Enlargement of the optic foramen and deformity of the sella turcica as seen by roentgenograms are suggestive but not diagnostic of this tumor and do not reliably indicate the extent of the lesion.

On the basis of our recent experience with 13 tumors in children under 12 years of age and information gained from review of the reported experience of others, it is felt that transfrontal craniotomy and orbital unroofing offers the best opportunity for satisfactory evaluation of the extent of these lesions, and the only opportunity for complete removal when this is surgically feasible. Total excision in 5 of our 13 cases is felt to have been accomplished.

When complete excision is inadvisable, histologic verification by biopsy is recommended, followed by roentgen therapy in the hope of slowing down further growth of the tumor. If an inoperable chiasmal tumor extends into the orbit and there is remaining vision, orbital decompression as well as irradiation is recommended.

REFERENCES

1. Ardouin, M., and Loisance, Y. Le diagnostic des agrandissements des canaux optiques. (A propos d'une tumeur du nerf optique propagée au chiasma). *J. Radiol. Électrol.*, 1950, *31:* 368–371.
2. Bailey, P., and Eisenhardt, L. Spongioblastomas of the brain. *J. comp. Neurol.*, 1932, *56:* 391–430.
3. Bakay, L., and White, J. C. Pneumoencephalography in chromophobe adenomas of the hypophysis. *J. Neurosurg.*, 1953, *10:* 284–296.
4. Bucy, P. C., Russell, J. R., and Whitsell, F. M. Surgical treatment of tumors of the optic nerve. Report of a case. *Arch. Ophthal., N. Y.*, 1950, s.2, *44:* 411–418.
5. Christensen, E., and Andersen, S. R. Primary tumours of the optic nerve and chiasm. *Acta Psychiat., Kbh.*, 1952, *27:* 5–16.
6. Cohen, I. Tumors of the intracranial portion of the optic nerve. *J. Mt. Sinai Hosp.*, 1951, *17:* 738–745.
7. Cushing, H. The chiasmal syndrome of primary optic atrophy and bitemporal field defects in adults with a normal sella turcica. *Arch. Ophthal., N. Y.*, 1930, *3:* 505–551; 704–735.
8. Dandy, W. E. Prechiasmal intracranial tumors of the optic nerves. *Amer. J. Ophthal.*, 1922, s.3, *5:* 169–188.
9. David, M., Brégeat, P., and Morice, J. La cisternographie, moyen d'étude du nerf optique intracranien et du chiasma. *Rev. neurol.*, 1951, *85:* 425–459.
10. Davis, F. A. Primary tumors of the optic nerve (a phenomenon of Recklinghausen's disease.) A clinical and pathologic study with a report of five cases and a review of the literature. *Arch. Ophthal., N. Y.*, 1940, s.2, *23:* 735–821; 957–1018.
11. Dubilier, W., Jr., von Gal, H., Freemond, A., and Evans, J. A. Orbital pneumotomography. *Radiology*, 1956, *66:* 387–392.
12. Duke-Elder, W. S. Text-book of ophthalmology. *St. Louis: C. V. Mosby Co.*, 1940, *3.*
13. Gluck, B. Glioma of the optic nerve. *Brit. J. Ophthal.*, 1932, *16:* 406–415.
14. Goalwin, H. A. One thousand optic canals. A clinical, anatomic and roentgenologic study. *J. Amer. med. Ass.*, 1927, *89:* 1745–1748.
15. Grinker, R. R. Tumors of the optic nerve. *Arch. Ophthal., N. Y.*, 1930, s.2, *4:* 497–508.

16. Grino, A., and Billet, E. The diagnosis of orbital tumors by angiography. *Amer. J. Ophthal.*, 1949, s.3, *32:* 897–911.
17. Hudson, A. C. Primary tumours of the optic nerve. *Roy. Ophthal. Hosp. Rep.*, 1912, *18:* 317–439.
18. Hudson, A. C., del Rio-Hortega, Wolff, E., Jefferson, G., Hird, B., and Neame, H. Discussion on tumours of the optic nerve. *Proc. roy. Soc. Med.*, 1940, *33:* 685–692.
19. Ingraham, F. D., Matson, D. D., and McLaurin, R. L. Cortisone and ACTH as an adjunct to the surgery of craniopharyngiomas. *New Engl. J. Med.*, 1952, *246:* 568–571.
20. Levitt, J. M. Primary glioma of the optic chiasm. Report of a case. *Amer. J. Ophthal.*, 1955, s.3, *40:* 856–861.
21. Love, J. G., and Benedict, W. L. Transcranial removal of intraorbital tumors. *J. Amer. med. Ass.*, 1945, *129:* 777–784.
22. Lysholm, E., and Olivecrona, H. On changes of the optic canals in cases of intracranial tumor. *Acta chir. scand.*, 1932, *72:* 197–209.
23. McFarland, P. E., and Eisenbeiss, J. Glioma of optic nerve. *Amer. J. Ophthal.*, 1950, s.3, *33:* 463–466.
24. McLean, J. M. Astrocytoma (true glioma) of the retina. Report of a case. *Arch. Ophthal., N. Y.*, 1937, s.2, *18:* 255–262.
25. Mannheimer, M. Glioma of the optic nerve. *Amer. J. Ophthal.*, 1946, s.3, *29:* 323–328.
26. Marshall, D. Glioma of the optic nerve as a manifestation of von Recklinghausen's disease. *Amer. J. Ophthal.*, 1954, s.3, *37:* 15–33.
27. Martin, P., and Cushing, H. Primary gliomas of the chiasm and optic nerves in their intracranial portion. *Arch. Ophthal., N. Y.*, 1923, *52:* 209–241.
28. Olivecrona, H. On suprasellar cholesteatomas. *Brain*, 1932, *55:* 122–134.
29. Pessagno, A. Studio stratigrafico assiale dell'orbita normale. *Ann. Ottalm.*, 1949, *75:* 210–215.
30. Posner, M., and Horrax, G. Tumor of the optic nerve. Long survival in three cases of intracranial tumor. *Arch. Ophthal., N. Y.*, 1948, s.2, *40:* 56–76.
31. Saebø, J. Primary tumour of the optic nerve (glioblastoma multiforme). *Brit. J. Ophthal.*, 1949, *33:* 701–708.
32. Scott, E., and Schmidt, F. F. Intradural tumors of optic nerve with report of a case. *Amer. J. Ophthal.*, 1920, s.3, *3:* 665–668.
33. Taveras, J. M., Mount, L. A., and Wood, E. H. The value of radiation therapy in the management of glioma of the optic nerves and chiasm. *Radiology*, 1956, *66:* 518–528.
34. Verhoeff, F. H. Primary intraneural tumors (gliomas) of the optic nerve. A histologic study of eleven cases, including a case showing cystic involvement of the optic disk, with demonstration of the origin of cytoid bodies of the retina and cavernous atrophy of the optic nerve. *Arch. Ophthal., N. Y.*, 1922, *51:* 120–140; 239–254.
35. Wilson, J. M., and Farmer, W. D. Glioma of the optic nerve. A critical review; report of two cases, with autopsy observations in one. *Arch. Ophthal., N. Y.*, 1940, s.2, *23:* 605–618.

OBSERVATIONS ON PATENCY OF CERVICAL CAROTID ARTERY FOLLOWING SURGICAL TREATMENT FOR THROMBOSIS*

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THE recent literature has contained some publications regarding the surgical treatment of thrombosis of the carotid artery in the neck. The reader gains the impression that the results have been quite encouraging. However, the follow-up has been extremely limited in value in assessing the usefulness of the various procedures because of the lack of any postoperative angiograms demonstrating restoration of circulation through the surgically treated thrombotic area. At the present time there are 4 surgical methods employed in the treatment of a thrombosed carotid artery in the neck. These are: 1) excision of the thrombosed segment and anastomosis of the severed ends of the artery, 2) excision of the thrombosed segment and replacement with an arterial or venous graft, 3) simple excision of the clot, and 4) thrombo-endarterectomy. Thrombo-endarterectomy seems to have gained the greatest popularity in the actual number of procedures carried out for this disorder. We wish to present 3 patients with symptoms caused by a thrombosed carotid artery in the neck proven by arteriography and surgically treated, with follow-up arteriograms and surgical explorations in 2 patients. All 3 patients improved following operation in the neck on the thrombosed artery—yet in 2 cases circulation had not re-established itself through the thrombosed segment.

The current interest in thrombosis of the carotid artery may have obtained its impetus from the surgical report of Webster *et al.*[7] in 1950 and from Fisher's[4] study of the pathology and frequency of this condition published in 1951. Webster *et al.*[7] had reported 4 cases of spontaneous thrombosis of the carotid arteries in the neck in which the thrombosed segments were removed and the vessels were ligated. One patient surprisingly improved following this procedure. Denman *et al.*,[2] in 1955, reported a patient in whom the thrombosed segment was excised and replaced with an arterial homograft. This would seem to be an excellent method, but the results have not been encouraging. Eastcott *et al.*,[3] in 1954, reported restoration of arterial continuity in a case in which direct anastomosis of the internal to the common carotid artery was done after excising a partially occluded carotid

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bifurcation and ligating the external carotid artery. However, there was no postoperative arteriogram, and the sole evidence of improvement was the relief of the patient's intermittent attacks of hemiplegia and absence of abnormal neurological findings. Lin *et al.*,[5] in 1956, reported the only successful restoration of circulation through the thrombotic area by means of excision of the bifurcation of the carotid artery and replacement by a saphenous vein graft. Three months later angiograms proved the graft to be patent. Arnstein[1] recently treated 10 patients by thrombo-endarterectomy. He stated that the simple excision of a thrombosed segment had been carried out by several different investigators and that he was not aware of any single reported patient being benefited following this procedure and that it had been tried in a sufficiently large number of cases by different surgeons. He also reported no success in anastomosing the common to the internal carotid artery after excising the thrombosed segment. However, some of the results following thrombo-endarterectomy were encouraging. He felt that the earlier a patient was operated upon the better the results would be. Again, no arteriographic or exploratory procedures were carried out to demonstrate adequate circulation through the previously thrombosed segment. Recently, Webster *et al.*[8] recorded 70 patients with thrombosed carotid arteries in the neck, of which 41 had an exploration and excision of the thrombosed vessel. In only 1 patient was there bleeding from the distal portion of the carotid artery after excision of the thrombosed segment, suggesting that the vessel might have been patent above the level of thrombosis. All patients showed evidence of atherosclerosis and none exhibited any canalization that appeared to be functioning. However, they gave no statistical analysis of the surgical results. Three of their patients were treated by reconstruction of the bifurcation after excision of the atheromatous segment and in each re-thrombosis occurred. They merely stressed the clinical picture, the ability to diagnose, and suggested the possibility of treatment by arterial graft. They hoped by excising the thrombosed segment, there would be no further emboli propagating from the thrombus.

Therefore, although the literature contains reports of surgically treated patients with this condition there is no adequate analysis of the results other than to say that there has been some improvement neurologically with cessation of attacks but no direct proof that the surgical procedure itself re-established circulation.

The purpose in presenting the following 3 cases is to suggest that the clinical improvement frequently observed after surgery on the thrombosed carotid artery in the neck may well be unrelated to the operative procedure as evidenced by our postoperative arteriograms and re-explorations.

CASE REPORTS

Case 1 (U.H. #9535-F). F.R., a 51-year-old white male, was admitted to the Neurosurgical Service of the John Sealy Hospital on Feb. 14, 1956 with the history of a sudden onset of hemiplegia and aphasia in September, 1949. He had had several

previous admissions since 1949, with a diagnosis of thrombosis of the left middle cerebral artery. Over the past few years speech and right-sided movement had improved. However, he had begun to experience seizures and headaches of progressive severity.

Examination revealed right spastic hemiparesis with a moderate degree of aphasia. On Feb. 17, 1956, pneumoencephalography showed a contracting lesion of the left cerebral hemisphere. On the same date, a left carotid arteriogram demonstrated thrombosis of the cervical portion of the left internal carotid artery. On Feb. 27, 1956 an arteriosclerotic plaque and clot were removed from the internal carotid artery and an anastomosis of the external carotid artery to the internal carotid artery above the thrombosed segment was done in order to attempt to re-establish circulation. The proximal portion of the internal carotid artery was ligated.

At the time of discharge from the hospital the patient felt improved. Objectively there was slight improvement of movement of the right hand and speech was slightly better.

The patient continued to improve. In order to determine the patency of the anastomosis, he was re-admitted 3 months later. On May 15, 1956, left percutaneous carotid arteriography was attempted but we were unable to puncture the artery. The vertebral artery was unintentionally entered. A week later, the left carotid artery was explored in the neck. A thin, cord-like, bloodless structure representing the remnants of the carotid artery was found. It should be pointed out that weak pulsations were felt preoperatively in the neck which in retrospect were caused by tugging of the aorta on the completely thrombosed carotid.

Comment. Certain important conclusions can be drawn from our observations on this patient. The aorta can tug sufficiently on a thrombosed, cord-like carotid artery to produce the effect of pulsations to the examiner. More important, the patient improved even though the vessel was completely thrombosed. After the anastomosis he improved further, suggesting that circulation had been re-established. However, exploration of this vessel 3 months later revealed only a solid cord of tissue, indicating that the surgical procedure had actually increased the degree of thrombosis which now involved the common, external and internal carotid vessels. In spite of this, the patient's clinical picture was improving, indicating an increasing development of an adequate collateral circulation from the opposite carotid artery. The left middle and anterior cerebral vessels were not visualized in the vertebral angiogram, suggesting that any collateral circulation must take place from the opposite carotid system. Had the re-exploration of the operative site been omitted, one would have felt that the anastomosis had contributed to the patient's improvement.

Case 2 (U.H. #2793-M). K.F.S., a 52-year-old white male, was admitted to the Neurosurgical Service of John Sealy Hospital in June, 1956, with a history of progressive weakness of the left upper extremity beginning 2 weeks before admission and then improving.

However, on admission the patient had weakness of his left hand and increased left deep tendon reflexes. Carotid arteriography revealed an incomplete thrombosis of the right internal carotid artery (Fig. 1). Several days later a thrombo-endarterec-

tomy was done. Postoperatively the paresis improved for 2 weeks followed by regression.

He was re-admitted on July 24, 1956, with weakness of the left leg and arm which was more pronounced than on the first admission. The following day, a carotid arteriogram showed complete obstruction of the right internal carotid artery at its origin (Fig. 2). Therefore, 2 days later, the right internal carotid artery in the neck was re-explored. At surgery the external carotid artery appeared somewhat larger than normal and was pulsating. The internal carotid artery appeared as a mere piece of scar tissue and did not pulsate. It is dubious as to whether the internal carotid artery could even have been identified if it were not for its relation to the external and common carotid arteries. The internal carotid artery was incised and a small thrombus was removed together with a considerable amount of calcified and degenerated intima. The entire length of the internal carotid artery in the neck was completely obliterated and, therefore, no anastomosis could be done. Postoperatively, this patient did show some improvement in his hemiparesis and even at this writing 7 months later, improvement is still evident.

Comment. In reviewing the second arteriogram, which showed complete occlusion of the internal carotid artery in the neck, filling of the carotid siphon intracranially was seen. The filling of the carotid siphon can be traced to the external carotid artery via the ophthalmic artery, as shown in Fig. 2.

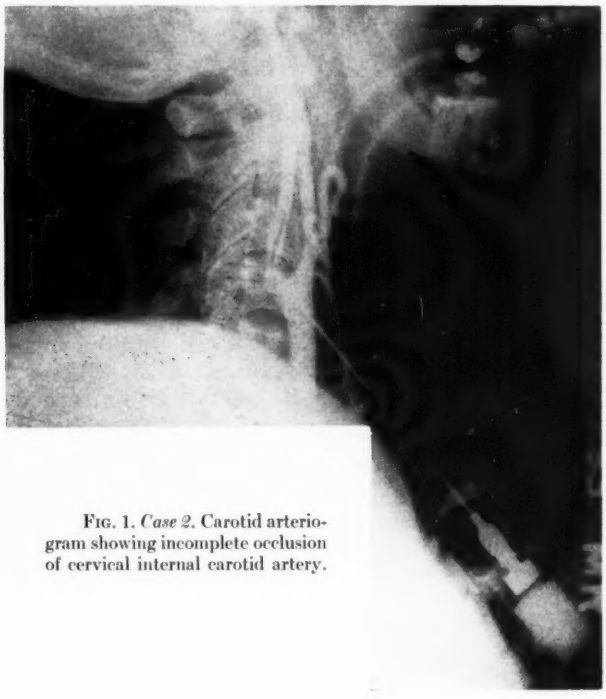

FIG. 1. *Case 2.* Carotid arteriogram showing incomplete occlusion of cervical internal carotid artery.

FIG. 2. *Case 2.* Arteriogram showing complete occlusion of cervical internal carotid artery and intracerebral blood flow through the collateral system consisting of the external carotid and ophthalmic arteries. (External carotid and ophthalmic arteries and intracranial carotid siphon are outlined in ink for better visualization in photograph.)

Therefore, this patient's improvement was not ascribable to the thrombo-endarterectomy but merely to the establishment of the collateral circulation of the external carotid artery and ophthalmic artery, as is well demonstrated. However, if the second exploration and arteriogram had been omitted with the patient continuing to improve as he has done, one would have felt that the initial thrombo-endarterectomy was beneficial.

Case 3 (U.H. #27168-M). O.A.H., a 46-year-old white male, was admitted on June 13, 1956, with a 2-week progressive loss of the use of his right upper extremity. Neurological examination revealed an increase in the right deep tendon reflexes and weakness of the right upper extremity. A left carotid arteriogram demonstrated complete obstruction of the internal carotid artery. On June 28, 1956, a thrombo-endarterectomy was performed. Postoperatively, the patient improved. No follow-up procedures have been done.

DISCUSSION

The surgical treatment of thrombosis of the carotid artery in the neck is relatively new and the impression that one gains from the literature is generally encouraging. Thrombo-endarterectomy has been most frequently employed. Another procedure used occasionally is an anastomosis of the external carotid into the internal carotid artery above the thrombosed

segment. The replacement of the thrombosed segments by arterial grafts has been advocated, though rarely done. From our perusal of the literature we find only 1 example of arteriography or re-exploration of the thrombosed areas after surgical repair.

Clinical improvement has been reported following thrombo-endarterectomy. Such was observed in our Case 2, although postoperative angiography and re-exploration revealed complete thrombosis. This patient's improvement was probably caused by an increased circulation to the affected hemisphere through the collateral supply of the external carotid and ophthalmic arteries and not through the site of the thrombo-endarterectomy. This is adequately demonstrated in Fig. 2 showing filling of the carotid siphon from the ophthalmic artery. This increase in collateral circulation was pointed out previously by Vaernet.[6] He described 4 patients with thrombosis of the carotid artery in the neck and collateral circulation through the external, ophthalmic and intracerebral vessels on the affected side. None of these patients was a surgical patient. These examples should certainly caution one against sacrificing the external carotid artery in patients with thrombosis of the internal carotid artery. One might well assume that the improvement of many of the patients subjected to thrombo-endarterectomy was actually caused by an increased collateral circulation via the external carotid artery and not by restoration of circulation through the site of the previous thrombosis. Of course, one is always aware of the fact that the collateral circulation can also come from the opposite carotid artery or the vertebral system.

In Case 1, the patient improved following the anastomosis of the external carotid artery to the internal carotid artery above the thrombosed segment, as has been reported by others. It should be noted that this patient had slowly improved over the years after his initial thrombosis but improved further following our surgical procedure. It, therefore, appeared that the patient had benefited from this anastomosis as evidenced by his improvement in speech and movements of the hand. However, the re-exploration demonstrated very clearly that circulation had not been re-established. Actually, the thrombosis was more extensive, involving the common and external carotid arteries; and obviously, the patient's improvement must have occurred from collateral circulation either from the opposite carotid artery or basilar system. However, in this instance it was probably not from the basilar artery, as evidenced by the vertebral angiogram, in which there was no filling of the anterior or middle cerebral arteries.

We wish to suggest that the previously reported improvements in patients with carotid artery thrombosis in the neck following a thrombo-endarterectomy or arterial shunt are the result of various collateral systems coming into play and are probably unrelated to the operation. However, this does not mean that further surgical attempts should be denied but that the procedure itself must be perfected and it must also be proven by post-operative angiograms or exploration that this segment is now functioning.

SUMMARY

Three patients are presented with thrombosis of the cervical portion of the internal carotid artery treated by thrombo-endarterectomy or anastomosis of the external carotid artery to the internal carotid artery above the thrombosis. All patients improved postoperatively. However, postoperative angiograms and surgical exploration revealed further thrombosis rather than re-establishment of circulation.

We wish to emphasize caution in the interpretation of favorable results.

REFERENCES

1. Arnstein, L. Surgical treatment. [Symposium on carotid artery thrombosis] *Proc. Congr. neurol. Surg.*, 1957, *3:* 156–165.
2. Denman, F. R., Ehni, G., and Duty, W. S. Insidious thrombotic occlusion of cervical carotid arteries, treated by arterial graft. A case report. *Surgery*, 1955, *38:* 569–577.
3. Eastcott, H. H. G., Pickering, G. W., and Rob, C. G. Reconstruction of internal carotid artery in a patient with intermittent attacks of hemiplegia. *Lancet*, 1954, *267:* 994–996.
4. Fisher, M. Occlusion of the internal carotid artery. *Arch. Neurol. Psychiat., Chicago*, 1951, *65:* 346–377.
5. Lin, P. M., Javid, H., and Doyle, E. J. Partial internal carotid artery occlusion treated by primary resection and vein graft. Report of a case. *J. Neurosurg.*, 1956, *13:* 650–655.
6. Vaernet, K. Collateral ophthalmic artery circulation in thrombotic carotid occlusion. *Neurology*, 1954, *4:* 605–611.
7. Webster, J. E., Dolgoff, S., and Gurdjian, E. S. Spontaneous thrombosis of the carotid arteries in the neck. Report of four cases. *Arch. Neurol. Psychiat., Chicago*, 1950, *63:* 942–953.
8. Webster, J. E., Gurdjian, E. S., and Martin, F. A. Carotid artery occlusion. *Neurology*, 1956, *6:* 491–502.

BETA RADIATION OF THE FELINE CAUDATE NUCLEUS: LATE RESULTS*

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CLINICAL stereotaxic neurosurgery, first introduced by Spiegel *et al.*[9] in 1947, and continuously modified by others, too numerous to mention, remains an unstandardized experimental procedure for want of a method for creating a lesion of predictable size and shape, let alone accuracy of placement. For relatively acute work in the laboratory, the use of stereotaxically implanted radioactive spheres, 0.86 mm. in diameter, met some of these requirements. The isotopes were Pd^{109} and Au^{198}. Therefore, a study of long-standing lesions was undertaken before applying the method on a clinical level, since others[1,8] have noted delayed effects after exposing the brains of laboratory animals to x-radiation, and the betatron beam. This report is restricted to a description of gross and microscopic changes 8 to 23 months following implantation of Au^{198} and Pd^{109} spheres, and evaluation of the method as an experimental neurosurgical adjunct. A method for accurate placement of the implanting trocar in the brain of man will be given in an additional communication.[7]

PHYSICAL DATA

Pd^{109} is a pure source of beta radiation. In contrast, Au^{198} emits both beta and gamma radiation, but is an excellent source of beta particles in the doses used in these experiments. The maximum energies of the beta particles emanating from both these sources are nearly identical, 0.95 and 0.97 mev. respectively. Approximate computations based on the strengths used in this study indicate that the gamma radiation of the Au^{198} spheres contributes a negligible fraction of the dose of radiation delivered within the area of tissue destruction. It was anticipated that the lesions would be discrete because of the limited penetration of beta particles in tissue. Adjustment of the size of the lesion was possible up to a point by increase in the strength of the source beyond the minimum for tissue destruction. However, the relation between these two quantities is not direct; it depends upon a number of factors such as beta ray attenuation, scattering, and the inverse square

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law. When a diameter of a lesion is required that exceeds the particle range of a certain pure beta emitter, it can best be obtained by employing an isotope that is a source of beta particles of greater maximum energy.[3,4] This, however, would not be the case with a source of both gamma and beta radiation, such as Au^{198}. For, when the strength of a mixed source is increased to the point that the gamma radiation becomes effective beyond the range of the beta particles, the diameter of the lesion will be increased roughly in accordance with the inverse square law. For this reason, it follows that gamma radiation will not produce a lesion as precisely demarcated as beta with its more limited range in tissue. It is this characteristic that makes a pure source of beta radiation the material of choice in stereotaxic neurosurgery. Additional physical factors, which must be taken into consideration in planning the volume of a lesion, are half life and self-absorption of beta particles within the source, both of which influence not only dose but rate of dose.[3,4]

EXPERIMENTAL DATA

In studying the late effects of beta radiation, a series of 12 cats were prepared. In 8, Au^{198} spheres, delivering from 0.5 to 2.05 mc., were placed in the caudate nucleus. Placements were unilateral on the right in 5; bilateral in 3. Survival time was varied from 305 days to 717 days. In 4 cats, unilateral placement of Pd^{109} spheres, delivering from 2.88 to 3.50 mc., was carried out. The survival time was 253 days in each animal.

All cats were observed at regular intervals. Those with Pd^{109} implantations showed no change in behavior or nutritional status. The same was true of the cats with unilateral Au^{198} placements. In the animals receiving bilateral implantations of Au^{198} there were transient and inconsistent neurological changes. These included piloerection, anisocoria and contraction of the nictitating membrane. The piloerection was maintained longest in a cat in which there was damage to the internal capsule. It was felt that these changes were probably caused by transient edema involving structures adjacent to the caudate nucleus or by injury to such regions in the insertion of the spheres.

All animals were killed under anesthesia by a method described elsewhere,[3,4] except for one, which died of intercurrent disease on the 471st day of the experiment. After thorough hardening of the brains in 10 per cent formalin, a single coronal section was made so as to expose the region of implantation for gross inspection. Following this, the brains were cut in thin coronal planes. Histological sections were prepared from one frontal and one occipital plane, from pons, medulla and cerebellum, as well as from the spinal cord. The entire region adjacent to the sphere was embedded and histological sections were prepared from many levels. Staining technics included hematoxylin and eosin, gallocyanin with and without van Geison's counterstain, Sudan black, Weil's method for myelin and Mallory's phosphotungstic acid-hematoxylin.

At gross inspection of the caudate nucleus, the lesions were seen as small,

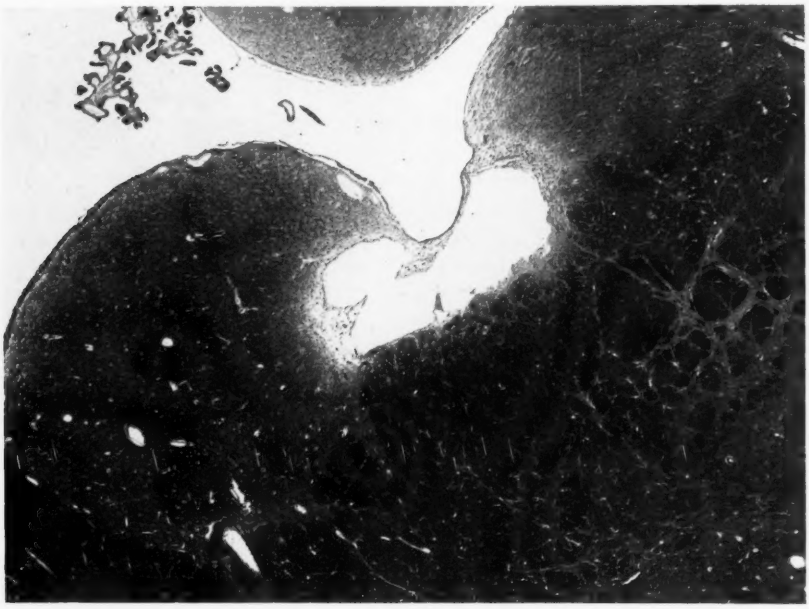

FIG. 1. Area of sphere placement in the caudate nucleus. Dosage 1.7 mc.; Au^{198}; 496 days. Weil's stain, ×18.

cystic areas with the sphere at the center. Further gross dissection of the brains showed no further abnormality except in one animal. This cat had an extensive bilateral symmetrical hydrocephalus, which was probably unrelated to the experimental procedure because the dose was small (0.5 mc.) and placement was unilateral.

Results with Au^{198} Spheres. In 7 animals, placement of the sphere was accurately within the substance of the caudate nucleus. The eighth animal was the cat with hydrocephalus, which may have altered the position of the caudate nucleus; this sphere was not recovered at autopsy. In some animals, there was a rim of tissue between the lesion produced and the ependyma medially (Fig. 1), and between the lesion and the internal capsule laterally. In others, the zone of tissue destruction reached and interrupted the ependymal surface (Fig. 2.) The lesion then appeared as a dimple, visible grossly.

In other respects, the zones about the spheres were very uniform, and can be described as a group. Each was roughly oval in outline and measured from 2.0 to 4.0 mm. in diameter as studied in histological sections after paraffin embedding. At the center, there was a cavity slightly larger than the sphere (which had been removed from the paraffin blocks). In this intervening region, there were delicate strands of collagenous tissue and rare capillaries arranged in a very loose-textured meshwork (Fig. 3). Astrocytes

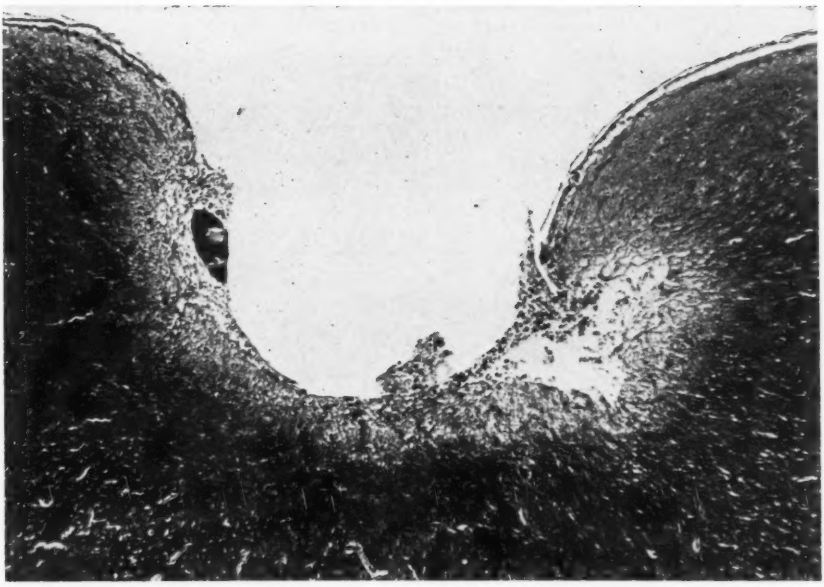

FIG. 2. Area of sphere placement in the caudate nucleus. In this animal, the zone of injury reached the ependyma. Dosage 1.7 mc.; Au^{198}; 496 days. Weil's stain, ×50.

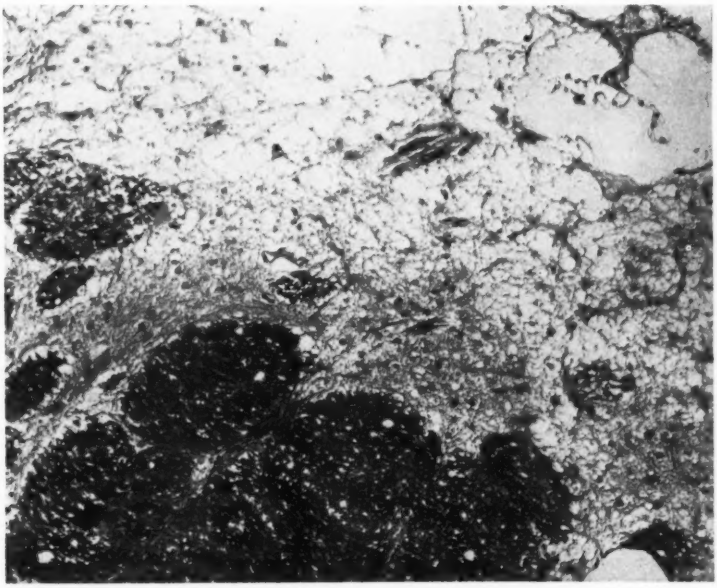

FIG. 3. Edge of area of sphere placement in the caudate nucleus. The delicate network, mostly collagenous, about the sphere is at the top. In the center is the astrocytic reaction in the surrounding tissue. Note the preservation of nerve fibers up to, and even into, this zone. Dosage 1.3 mc.; Au^{198}; 471 days. Weil's stain, ×175.

in this zone were few. In one cat, a small amount of phagocytosed hemosiderin was also found.

External to this layer, and forming the wall of the cavity, was a close-textured mat composed almost entirely of astrocytes (Fig. 3). These cells had the usual stellate processes and vesicular nuclei of adult forms. The astrocytic layer was nowhere wider than half the diameter of a high-power microscopic field. Immediately beneath, and even jutting into the layer, there were bundles of nerve fibers which were completely preserved in morphological detail (Fig. 3). The myelin sheaths stained deeply and uniformly with Weil's method; no vacuoles were seen. The transition was strikingly abrupt from entirely destroyed tissue to tissue wholly normal in morphology. Occasionally, the staining of the myelinated fibers of the internal capsule nearest to the lesion was a little paler than elsewhere as seen at low power, but on high-power study, the details of the structure of the myelin sheaths appeared well preserved. No evidence of pressure atrophy was found.

Microscopic sections of cerebral cortex and of cerebellum showed no lesions. Especial attention was given to the region of the pyramidal tract in sections of pons and medulla. If there had been extension of necrosis or reaction to the internal capsule, it was felt that retrograde degenerations might be apparent at these levels. No such changes were present, further evidence that the fibers of the internal capsule remained intact.

Results with Pd^{109} Spheres. Qualitatively, the response to the Pd^{109} spheres was identical in detail with that of the Au^{198} spheres (Fig. 4). The lesions were very slightly larger, varying from 3.0 to 5.0 mm. in diameter. They extended to the internal capsule, but involved few of its fibers in any animal. In sections of the pons and medulla, there were very occasional degenerated nerve fibers in the pyramidal tracts, but these were more likely the result of the placement incision than of involvement of the internal capsule by radiation reaction. The incision could be traced in some animals across the upper part of the anterior limb of the internal capsule.

DISCUSSION

Cairns and Fulton,[5] 1930, were probably the first investigators to avail themselves of progressive destruction brought about by ionizing radiation, when they placed radon seeds in juxtaposition to the dorsal spinal cord of laboratory animals, and observed the succession of physiological changes. Borison and Wang,[2] in 1951, destroyed the canine vomiting center by free-hand placement of radon seeds in the medulla. These authors attributed their success in this sensitive area to progressive development of the lesion up to a calculated limit. Their dosage was determined by correlation of volume of tissue destroyed, and the amount of radiant energy evolved by the source. More recently, Grossman and Wang[6] have applied a modification of this stereotaxic technic in a physiological study.

For purposes of the present study and others already completed,[3,4] a technic was evolved for stereotaxic placement of calibrated sources of radio-

FIG. 4. Edge of area of sphere placement in the caudate nucleus in a cat receiving 2.88 mc. from Pd^{109} source. Note the similarity of the tissue reaction to that induced by Au^{198} (Fig. 3). Survival 253 days. Weil's stain, ×175.

activity. Glass and gold radon seeds, which were first employed, had the disadvantage of bulk. This problem was surmounted by using artificially produced radioactive isotopes delivered as spheres 0.86 mm. in diameter. At the same time, a marked advantage was gained through selection of half life and type of radiation. The volume and shape of lesions produced by this approximation of a point source were predictable and the perimeters were discrete. Because the resulting necrotizing effect was gradual, an additional advantage accrued.

Lesions of greater volume resulting from radionecrosis are better tolerated than smaller ones created by more abrupt methods.[3,4] The effects produced by the Pd^{109} spheres were associated with a more rapid rate of dose than was delivered by Au^{198} spheres. Thus in short-term experiments,[3,4] more severe tissue reaction resulted from Pd^{109}, even though lower doses in millicurie hours were used. So, too, in the long-term experiments of the present series, the area of residual damage with Pd^{109} was a little larger than with Au^{198}.

In acute lesions, the transition zone from lesions to normal structures was irregular, but in the long-term experiments, the zone of reaction was much narrower and more regular with nerve fibers preserved up to a thin, astrocytic layer (Figs. 3 and 4).

Since the lesions in the animals of this series appeared very similar at the

various intervals after operation (253 to 717 days), it was felt unlikely that still longer intervals would reveal further changes or extensions of the tissue reaction.

Examination of many areas of the brain other than the caudate nucleus showed no changes related directly to the radiation. It was felt that the approximation of a point source by the use of small spheres was an important factor in the sharp localization of tissue reaction.

SUMMARY

The late effects of beta radiation in the caudate nucleus were studied after stereotaxic placement of Au^{198} and Pd^{109} spheres.

Animals were allowed to survive from 253 to 717 days.

Small, sharply localized lesions resulted without changes in cerebral structures other than the caudate nucleus.

The approximation of a point source for administering beta radiation was felt to be an important factor in the localization of tissue reaction.

REFERENCES

1. Arnold, A., Bailey, P., and Laughlin, J. S. Effects of betatron radiations on the brain of primates. *Neurology*, 1954, *4:* 165–178.
2. Borison, H. L., and Wang, S. C. Quantitative effects of radon implanted in the medulla oblongata: a technique for producing discrete lesions. *J. Comp. Neurol.*, 1951, *94:* 33–55.
3. Boyesen, S., and Campbell, J. B. Stereotaxic implantation of calibrated Pd^{109} and Y^{90} spheres: a technique for producing predictable subcortical lesions in the brains of laboratory animals. *Yale J. Biol. Med.*, 1955–1956, *28:* 216–224.
4. Boyesen, S., and Campbell, J. B. Stereotaxic implantation of artificially produced radioactive isotopes: a technique for producing quantitated subcortical lesions in the brains of laboratory animals. *J. comp. Neurol.* (In press.)
5. Cairns, H., and Fulton, J. F. Experimental observations on the action of radon on the spinal cord. *Lancet*, 1930, *2:* 16–18.
6. Grossman, R. G., and Wang, S. C. Diencephalic mechanism of control of the urinary bladder of the cat. *Yale J. Biol. Med.*, 1955–1956, *28:* 285–297.
7. Rossi, H. H., and Campbell, J. B. In preparation.
8. Russell, D. S., Wilson, C. W., and Tansley, K. Experimental radio-necrosis of the brain in rabbits. *J. Neurol. Neurosurg. Psychiat.*, 1949, n.s. *12:* 187–195.
9. Spiegel, E. A., Wycis, H. T., Marks, M., and Lee, A. J. Stereotaxic apparatus for operations on the human brain. *Science*, 1947, *106:* 349–350.

VERTEBRAL ARTERIOGRAPHY IN THE STUDY OF SUBARACHNOID HEMORRHAGE

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THE understanding and definitive treatment of subarachnoid hemorrhage must necessarily develop with the evolution of effective methods of investigating the cerebral vascular tree. Bilateral percutaneous carotid arteriography has become almost routine practice in the study of subarachnoid hemorrhage. This technique has shown that aneurysms, angiomas, and, very rarely, tumors are responsible for a large percentage of hemorrhages. A significant group remains in which no source for bleeding is found.

The purpose of this paper is to report the additional information gained by vertebral arteriography.

This series is composed of 60 cases of spontaneous subarachnoid hemorrhage in which bilateral carotid arteriography failed to reveal a source of bleeding. No cases were included in which a posterior lesion was suspected and diagnosed by vertebral arteriography as a primary procedure. The diagnosis was confirmed by lumbar puncture in all cases. In no case was there an apparent hemorrhagic diathesis. None of the patients had suffered craniocerebral injury as part of his illness, so that traumatic subarachnoid hemorrhage is not germane to this group.

Vertebral arteriography was entirely percutaneous via an anterior approach. Difficulties of technique have been largely overcome since adoption of the Sheldon needle.[7,8] General endotracheal anesthesia was used in the great majority of cases to spare the patient the pain of inadvertent nerve-root irritation by the needle (carotid angiography is routinely performed under local anesthesia).

Radiological Findings. In 16 cases (26 per cent) vertebral arteriography demonstrated a lesion capable of producing and assumed to be responsible for the subarachnoid hemorrhage. Eight of these were aneurysms and 8 were angiomas. An analysis of the findings is given in Table 1.

The posterior cerebral arteries were demonstrated by carotid injection in 21 of the 60 cases (35 per cent), a considerably higher number than the 20 per cent reported by Moniz.[4] Analysis of the 16 pathological cases showed no filling of the posterior cerebral arteries in 13. Both posterior cerebral arteries were demonstrated in 1 case, and one posterior cerebral artery was filled in 2 cases. In none of the last 3 was the "feeding" or source vessel demonstrated.

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TABLE 1

Analysis of 16 pathological cases

I. *Aneurysms*—8 cases
- A. Basilar bifurcation—3 cases
- B. Posterior cerebral—4 cases
  - all near basilar bifurcation
    - Left—3
    - Right—1
- C. Vertebral—1 case
  - left vertebral artery at level of foramen magnum (Figs. 1 and 2)

II. *Angiomas*—8 cases
- A. Supratentorial—2 cases
  1. inferomedial surface of left occipital lobe (Fig. 3)
  2. posterior portion of right temporal region
- B. Supra- and infratentorial—1 case
  3. mainly in distribution of right posterior cerebral artery
- C. Infratentorial—5 cases

Midline

4. limited to vermis of cerebellum
5. pontine-filling mainly from right posterior cerebral artery
6. filled by both posterior cerebral arteries

Lateral

7. superior surface of right cerebellar hemisphere
8. left cerebellum-filling mainly from left superior cerebellar artery and left posterior inferior cerebellar artery

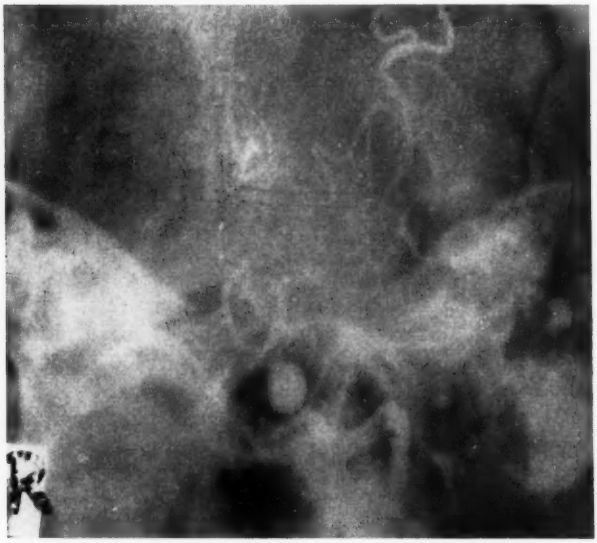

FIG. 1. Left anteroposterior vertebral arteriogram showing aneurysm.

FIG. 2. Same case as in Fig. 1. Postoperative left vertebral injection showing occlusion of the artery with slight retrograde thrombosis.

The plain roentgenograms of the skull were normal in all but 1 case, in which erosion of the dorsum sellae was seen—a sign of no localizing value.

Clinical Findings. The series was comprised of 32 males and 28 females, ranging in age from 6 years to 66 years. Seven patients (11.5 per cent) were under 20 years of age; 36 (60 per cent) were between 20 and 50; and 17 (28 per cent) were over 50 years of age.

FIG. 3. Left lateral vertebral arteriogram showing anterior occipital angioma.

Nine (15 per cent) of the patients exhibited hypertension as judged by several recordings of diastolic pressure over 90 mm. of mercury.

Of the group, 37 (61.5 per cent) retained consciousness while 23 patients lost consciousness, either initially or early in the course of their illness.

In every case save one, the main symptom was severe headache, usually of abrupt onset. The physical signs were of two types: those caused by contamination of the subarachnoid space with blood, and the signs of focal neurological deficit. Stupor, stiff neck, Kernig's sign, i.e., the manifestations of meningeal irritation, were reported in some degree in all cases. Focal neurological abnormality was present in 15 patients (25 per cent), in 7 (11 per cent) of whom total arteriography had revealed no lesion. Eight (50 per cent) of the pathological group had physical signs. Hemiparesis, hemianopia, facial weakness and ocular nerve palsy (mainly 3rd and 6th nerves) were common to both groups, as was the occasional finding of retinal hemorrhages.

DISCUSSION

The number of angiomas and aneurysms discovered by vertebral arteriography was surprisingly high. Walton,[10] summarizing many published reports including postmortem material, felt that 15 per cent of aneurysms were on the vertebral-basilar system. McKissock and Walsh,[2] and Falconer[1] reported posterior aneurysms as occurring in 3 to 6 per cent of their clinical series. Angiomas of the brain stem and cerebellum have been considered quite uncommon.[3,6,10]

It was further thought by Olivecrona and Riives[5] that since the majority of angiomas received part of their blood supply from the internal carotid artery, they would be revealed by carotid arteriography. In the present series, despite a high percentage of cases in which there was filling of the posterior cerebral artery from the internal carotid artery (35 per cent), none of the posterior lesions was demonstrated by carotid arteriography.

Few have been optimistic about the surgical treatment of posterior vascular lesions. Yet, 3 of the 16 patients were cured by total excision of the lesion: occipital angioma, vermis angioma and vertebral aneurysms (Mr. W. McKissock, Mr. V. Logue).

Many physicians have felt that those patients who survived their original ictus and had no lesion demonstrated by arteriography, had a far better prognosis than those in whom a lesion was shown. Walsh[9] has recently published data to support this belief. We subscribe to this theory albeit the follow-up of the present series is not long enough to be valid statistically.

CONCLUSIONS

Vertebral arteriography should be undertaken when bilateral carotid arteriography fails to show a source for subarachnoid hemorrhage.

Vertebral arteriography may show a surgically accessible lesion. If total study is negative, the physician can more securely offer the patient a good prognosis.

Both posterior cerebral arteries must be filled to exclude supratentorial angiomas and aneurysms.

SUMMARY

1. Sixty cases of subarachnoid hemorrhage with negative carotid arteriograms have been investigated with vertebral arteriography.

2. The radiological findings have been detailed.

3. A résumé of the major clinical findings has been presented.

We wish to thank Mr. Wylie McKissock for allowing us to analyze his cases of subarachnoid hemorrhage. A few of the patients were under the care of Mr. Logue and Dr. Carmichael, whom we also wish to thank.

REFERENCES

1. Falconer, M. A. Surgical pathology of spontaneous intracranial haemorrhage due to aneurysms and arteriovenous malformations. *Proc. roy. Soc. Med.*, 1954, *47:* 693–700.
2. McKissock, W., and Walsh, L. Subarachnoid haemorrhage due to intracranial aneurysms. Results of treatment of 249 verified cases. *Brit. med. J.*, 1956, *2:* 559–565.
3. Martin, J. P. Discussion on some less common cerebrovascular diseases. *Proc. roy. Soc. Med.*, 1951, *44:* 845–847.
4. Moniz, E. L'angiographie cérébrale. Ses applications et résultats en anatomie, physiologie et clinique. *Paris: Masson & Cie*, 1934, 327 pp.
5. Olivecrona, H., and Rives, J. Arteriovenous aneurysms of the brain. Their diagnosis and treatment. *Arch. Neurol. Psychiat., Chicago*, 1948, *59:* 567–602.
6. Richardson, J. C., and Bagnall, A. W. Recurrent subarachnoid haemorrhage due to an arterial angioma of the cerebellum and brain-stem. *Canad. med. Ass. J.*, 1940, *43:* 111–115.
7. Sheldon, P. A special needle for percutaneous vertebral angiography. *Brit. J. Radiol.*, 1956, *29:* 231–232.
8. Swann, F. G. Personal communication.
9. Walsh, L. S. Subarachnoid haemorrhage. *Acta radiol., Stockh.*, 1956, *46:* 321–325.
10. Walton, J. N. Subarachnoid haemorrhage. *Edinburgh & London: E. & S. Livingstone*, 1956, xv, 350 pp.

SUCCESSFUL SEPARATION OF CRANIOPAGUS TWINS

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THE first mention of craniopagus twins appears to be in the *Cosmographiae Universalis* of Sebastian Münster.[6] The Latin (1559), French (1575), and German (1628) editions all include a brief reference to the birth of two daughters joined at the foreheads, in Bierstadt in September 1495. There are wood cuts in both the Latin and German editions showing two girls standing with their heads turned toward each other and united, the right frontal region of one to the left frontal region of the other. The most astounding feature of this report is that these two children lived to the age of 10, when one died and was then "cut off" from the other. The second child then "died soon after her sister." This was not only the first case reported and the first recorded attempt at separation but also established a record for longevity.

Trenkner[12] in 1938 collected 24 cases from the literature and reported an additional case. However, he did not mention the cases of Löwenberg,[5] Dabelow,[3] Rogers,[8] Schneider,[9] and Sonnenburg[10] or the 6 cases of Baudouin.[2] Since Trenkner's report, Robertson,[7] Barbosa,[1] and Sugar and his associates[4,11] have reported additional cases. The case reported here would then be the 40th. This does not take into account Warschauer's[13] cases. This author reported a case in 1909 and referred to 21 cases from the literature. Only 2 are identified by an author. These 2 and the case reported by Warschauer are included by Trenkner. How many of the remaining 19 have been included in other reports is not clear.

The attachment or point of union of the heads is quite variable and often is not described accurately. However, a parietal union appears most common. Varying degrees of rotation of the children and varying states of fusion of the dura mater, absence of the dura mater and fusion or continuity of the brains are reported.

A number of attempts (6) at surgical separation are recorded, usually in the earlier cases after the death of one child. In the case reported by Robertson[7] an exploration was carried out at 4 months of age but separation was not performed. Until the case of Sugar and his associates[4,11] there were no recorded survivals of more than a few hours after separation. One of Sugar's patients died a month after operation without regaining consciousness, the other survived. In the case to be reported both children have survived and

are well at the age of 2 years. One has some neurological deficit, the other appears to be developing normally.

These twin girls were born in a Chicago hospital by Caesarian section on Oct. 1, 1954. They were transferred to Mercy Hospital 45 days later on Nov. 15, 1954. At that time their combined weight was 15 pounds, 12 ounces. Both girls appeared to be healthy normal children, except for the union of the two heads. Figs. 1 and 2 show the location and extent of this union and Figs. 3 and 4 show the roentgenographic appearance of the union. It was felt initially that the manner of junction with the two heads nearly at an angle of 180° indicated that the contents of the intracranial cavities were separate and especially that each child had a separate longitudinal sinus. It was impractical for either child to lie on her back. The two heads were not in the same anteroposterior plane and this necessarily produced some lateral rotation of the cervical spine in each child as they were placed first on one side or the other. This rotation of the cervical spine was to result in torticollis for the smaller child.

FIG. 1. Photograph of children at 2 months of age. L. faces the camera.

FIG. 2. Photograph of children at 2 months of age. S. faces the camera.

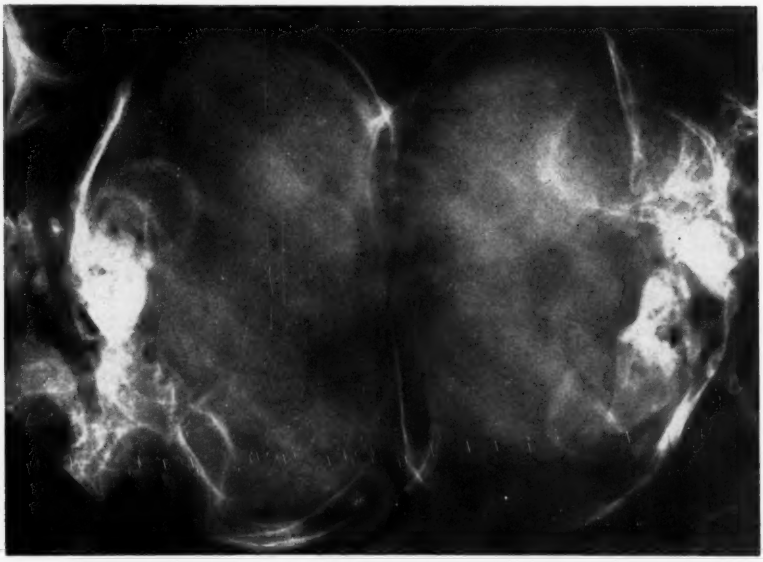


FIG. 3. Lateral roentgenogram of skulls at 2 months of age. L. faces down on the left.

FIG. 4. Anteroposterior roentgenogram of skulls at 2 months of age. L. is on the left.

While it was not feasible to weigh the children separately, one child was obviously larger from the onset. She will be referred to as L. and her sister as S. This designation should serve to differentiate them clearly and avoid the use of the initials of their names. When they were 8 days old, their combined weight was 12 pounds, $13\frac{1}{2}$ ounces. The circumference of the head of L. was 36.5 cm., that of S. was 35 cm.

TABLE 1

Comparative blood counts of L. and S.

Examination of Blood	November 29, 1954		April 8, 1955	
	L.	S.	L.	S.
Color index	.99	.98	.90	.90
Red blood cells	3,500,000	3,850,000	3,700,000	3,640,000
Grams hemoglobin	10.9	11.7	10.4	10.2
Per cent hemoglobin	69.8	74.9	66.6	65.3
White blood cells	12,800	13,000	13,200	12,650
Total polymorphonuclear leucocytes	18	16	31	25
Eosinophils	2	3	5	7
Lymphocytes	68	70	59	52
Monocytes	12	10	10	10

When more complete measurements were carried out, 45 days after birth, the circumference of the head of L. was 39 cm., that of her chest 36.5 cm., and her crown-rump length was 35 cm. The circumference of the head of S. was 37 cm., that of her chest 32 cm., and her crown-rump length was 33 cm. The right parietal of L. and the left parietal boss of S. were more prominent than their counterparts. The occiput of S. was asymmetrical, the left side being much more prominent. The blood counts of the two children were never identical but, on the other hand, did not vary significantly. Table 1 gives the data for two sample counts on each child on the same dates.

The children grew slowly. The circumference of the union slowly increased from the initial 31 cm. on Nov. 15, 1954 to 34 cm. on April 2, 1955. Table 2 shows the progressive changes in the combined weights and individual measurements of the children from Nov. 15, 1954 to April 2, 1955 ($4\frac{1}{2}$ months). Electroencephalography was performed simultaneously on the children on Dec. 11, 1954. This revealed an essentially normal cortical rhythm for each child that was not synchronous for the two children.

A pneumoencephalogram was attempted on L. on Jan. 18, 1955. With the child in the left lateral position and the table tilted with her head up and her sister lying

TABLE 2

Development chart of L. and S.

Date	Combined Weight	Head Circumference		Chest Circumference		Crown-Rump	
		L. cm.	S. cm.	L. cm.	S. cm.	L. cm.	S. cm.
10- 8-54	12 lbs. 13½ oz.	36.5	35				
10-15-54	13 lbs. 4½ oz.	36.8	35.6				
11-15-54	15 lbs. 12 oz.	39	37	36.5	32	35	33
11-29-54	16 lbs.	39	37	38	35.5		
12-10-54	16 lbs. 1 oz.	39	37.5	38.5	35.5	37.5	33.5
12-24-54	16 lbs.	39	37.5	38.5	35.5	37.3	33.5
1-10-55	15 lbs. 12 oz.	39	38	38.5	35.5	37.5	34
1-28-55	16 lbs. 10 oz.	40.5	39	38.5	35.5	37.5	35
3- 7-55	17 lbs. 10 oz.	41.5	40	39	36	41.5	36
4- 2-55	18 lbs. 14 oz.	42	40.5	39	36	43	40

above her, a spinal puncture was carried out at the 3rd lumbar interspace. Only about 20 cc. of fluid were obtained in spite of the substitution of air. Roentgenograms were then taken but no air had entered the ventricular system. Air could be seen in the basilar cisterns and over the right cerebral hemisphere. The same procedure was carried out on S. 4 days later on Jan. 22, 1955. Again only 20 cc. of fluid could be obtained and again the roentgenograms of the skull showed air only in the basilar cisterns and the right cerebral subarachnoid spaces.

Simultaneous ventriculography was carried out under general anesthesia on Feb. 17, 1955. With the children in the right lateral position and a separate surgical team for each child, burr holes were made in each left parieto-occipital region through small vertical scalp incisions. The left lateral ventricle of L. was easily entered, about 15 cc. of fluid were removed and air was substituted. It was more difficult to enter the ventricle of S. and only 5 cc. of fluid were obtained. However, roentgenograms demonstrated satisfactory filling of the ventricles of each child. Fig. 5 is a roentgenogram showing a lateral view of the ventricles of both children. Fig. 6 is an anteroposterior view of the ventricles of L. Fig. 7 is a similar projection of the ventricles of S. The ventricles of L. are essentially normal except that there is moderate symmetrical enlargement of the posterior horns of the lateral ventricles. The ventricles of S. are somewhat dilated and there is a localized enlargement of the temporal horn of the right lateral ventricle. There were several localized collections of air in the Sylvian fissure and over the vertex. It was felt that the ventriculogram of S. was abnormal but that the abnormalities seen were not sufficient to preclude normal development.

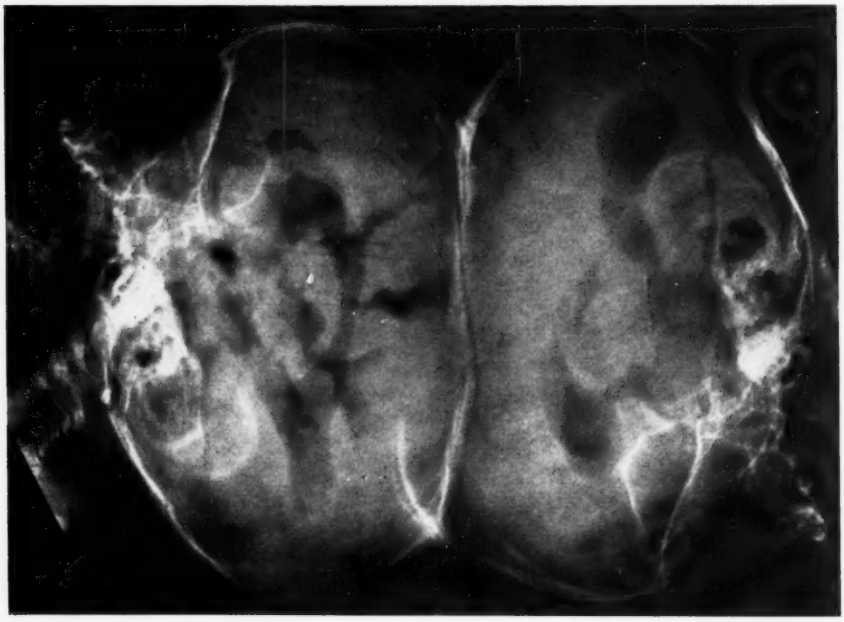
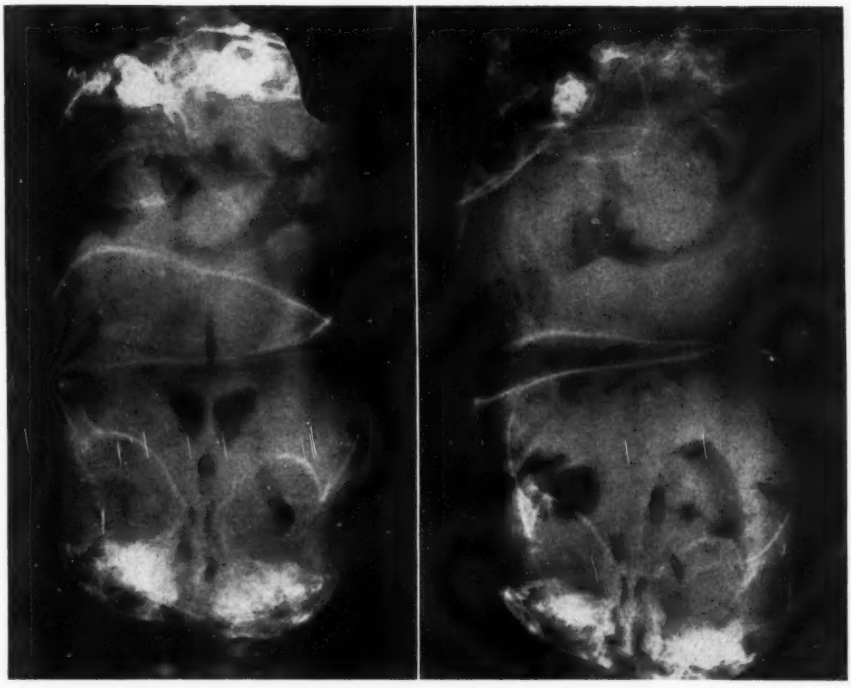


FIG. 5. Lateral projection of ventriculograms. L. faces up on the left.



FIGS. 6 and 7. Anteroposterior ventriculograms of L. (*left*) and S. (*right*).

The next and most important step in the pre-operative studies was to visualize the circulation of the two brains and particularly to demonstrate, if possible, the individual longitudinal sinus that each child was thought to possess. Right simultaneous carotid angiography was carried out under general anesthesia on Feb. 24, 1955. Two surgical teams, one for each child, exposed the right carotid vessels and a polythene catheter was introduced into the right internal carotid artery of each child. The children were then placed with their heads on the Sanchez-Perez seriograph and successive injections of 7 cc. of Hypaque were made simultaneously into each carotid artery while serial roentgenograms were taken. It was not possible to make satisfactory anteroposterior projections but satisfactory lateral films in both arterial and venous phases of filling were obtained. Fig. 8 shows a lateral roentgenogram of the arterial phase in the two children, Fig. 9 is a lateral roentgenogram of the venous phase. The carotid arteries and their main branches are filled in each child. There is better filling of the peripheral cerebral vessels in S. In the venous phase, two longitudinal sinuses can be seen. There is an apparent communication near the vertex, apparently about 5 mm. in diameter. In L. most of the cerebral veins drain into dural venous sinuses at the base or into the internal cerebral veins. The few superior cerebral veins are mostly in the posterior part of the intracranial cavity. The superior longitudinal sinus did not appear important to the venous drainage of the brain of L.

FIG. 8. Lateral roentgenograms of arterial phase of angiogram. L. faces up on the left.

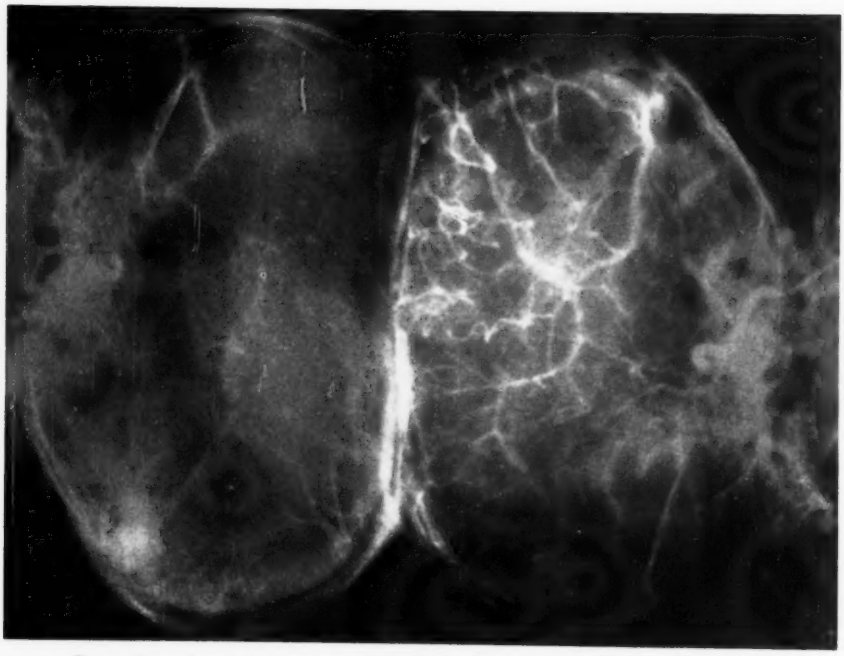


FIG. 9. Lateral roentgenogram of venous phase of angiogram. L. faces up on the left.

It was now decided that surgical separation of the children was feasible.

Operations. The plastic surgical team carried out elevation of a scalp flap from the right parietal region of S. under general anesthesia on Mar. 8, 1955. This flap had its base at the union and obtained its blood supply from the scalp of L. Fig. 10 is a diagram showing the relationship of this flap. After it had been reflected a skin graft, .005 inch thick, was taken from the abdomen of L. and placed on the under aspect of this flap and the denuded parietotemporal region of S. The flap was then resutured.

On Apr. 5, 1955, again under general anesthesia, a similar procedure was carried out on the other side. This flap was elevated from the left parietal region of L.

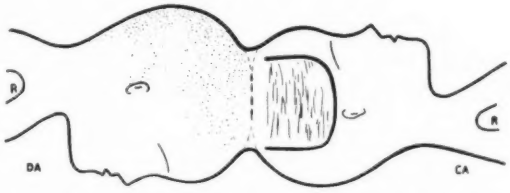


FIG. 10. Diagram of scalp flap in right parietal region of S. L. faces down on the left.

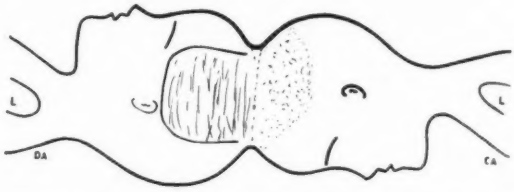


FIG. 11. Diagram of scalp flap in left parietal region of L. S. faces down on the right.

The base of the flap was again at the union with the blood supply derived from the scalp of S. Fig. 11 is a diagram illustrating this flap. The skin graft was taken this time from the abdomen of S. These wounds including the donor sites all healed satisfactorily. The condition of the children appeared satisfactory and their combined weight was now 20 pounds.

The actual surgical separation was carried out on Apr. 21, 1955, again under general anesthesia. The children were placed in the left lateral position. Both heads were prepared surgically and draped so that the children could be turned without contaminating the operative field. The scalp flap was reflected from the right parietotemporal region of S. and the skin graft on its under surface was sectioned along the line of the bony union. A burr hole was made at the posterior end of the union with reference to S. and the anterior end with reference to L. This revealed a paper-thin sheet of bone between the two heads. This was found to extend toward the vertex for about 3 cm. Later a similar thin sheet of bone was found to extend inward between the two brains at the other end of the union for about the same distance. No such bone extended in from either parietal region. An initial effort was made to preserve this thin bone at either end but because of difficulty in retraction it was eventually sacrificed everywhere. Where it was present a well developed dura mater was present on each side and the separation of dura mater from this bone was easy. Where bone was not present splitting of the dura mater was tedious and at times it was diffi-

cult to maintain a consistent plane of cleavage, especially in this right parietal region where the dura mater proved to be very thin. After the dura mater had been separated up to the midline on the right side the children were turned into the right lateral position. The dissection was more tedious and difficult on the left side but eventually it was complete everywhere except for an area in the midline at the junction of the anterior and middle thirds of the longitudinal sinus of L. and correspondingly the posterior and middle thirds of the sinus of S. Here severe bleeding was encountered and it was apparent that this was the point of communication between the two sinuses that had been visualized in the venogram. At this point, there was difficulty with the airway of L. and the final separation of the dura mater was accomplished rapidly. The opening into each longitudinal sinus was covered with gelatin sponge and physical separation of the children was complete, 4 hours and 15 minutes after the operation had begun.

The children were now on separate operating tables and resuscitation of L. was accomplished rapidly. Two plastic surgical teams now took over the operation. Closure of the wound of L. was effected rapidly. A cottonoid pack was left in place over the gelatin sponge on the opening in the longitidinal sinus. The scalp flap was sutured loosely to the dura mater, a head dressing was applied and the child was returned to the ward. Fig. 12 is a diagram showing how the scalp flap covered the right side of the bone defect and extended over the midline but failed to cover the entire area of exposed dura mater.

A more definitive closure was carried out on S. The skin graft was removed from the under surface of her scalp flap and then this flap was stretched over the dura mater as far as possible before suturing it in place. The skin graft which had been removed from the under surface of the flap was used to cover the remaining dura mater. Then, a pressure dressing was applied and this child was returned to the ward. She was awake, crying and in good condition. Fig. 13 is a diagram showing the relation of the scalp flap to the skull defect in this child.

Postoperative Course. L. soon rallied after her return to the ward. Two days later the dressing was removed and the cottonoid pack over the longitudinal sinus was removed. The scalp flap was sutured further, stretching it as much as possible. The bony defect measured 9.5 cm. in the anteroposterior diameter and 9 cm. in the transverse diameter. The area of exposed dura mater measured 9 cm. in the anteroposterior diameter and 4 cm. in the greatest transverse diameter.

The condition of the wound and the general condition of the child remained satisfactory. By May 12, 1955 the exposed dura mater was covered with granulation tissue and on this date a skin graft, approximately .009 inch thick, was removed with a Padgett dermatome from the upper abdomen and lower chest and sutured over the exposed dura mater. This graft healed satisfactorily although a month later a small arachnoid cyst had to be resected from the junction of the skin graft and scalp flap in the right frontal region. Mild bulging of the scalp flap gradually developed, indicating some weakness of the dura mater on the right side of the skull defect.

This child was dismissed from the hospital on July 14, 1955. At that time she was 9½ months old and weighed 14 pounds. The circumference of both the head and chest was 42.5 cm. Figs. 14 and 15 are anteroposterior and lateral roentgenograms of the skull showing the extent of the bony defect. Because of recurrence of the small subarachnoid cyst in the right frontal region with occasional drainage of cerebrospinal fluid, L. was re-admitted to the hospital Aug. 26, 1955 and the next day the cyst was resected and a plastic closure was carried out. This healed without incidence and the child left the hospital on Sept. 11, 1955.

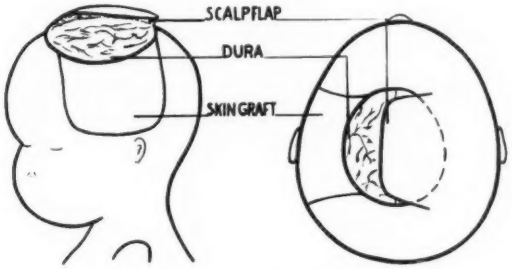


FIG. 12. Diagram of closure on L.

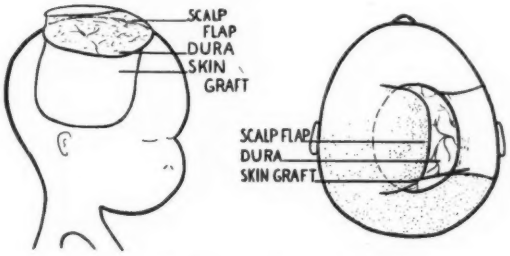
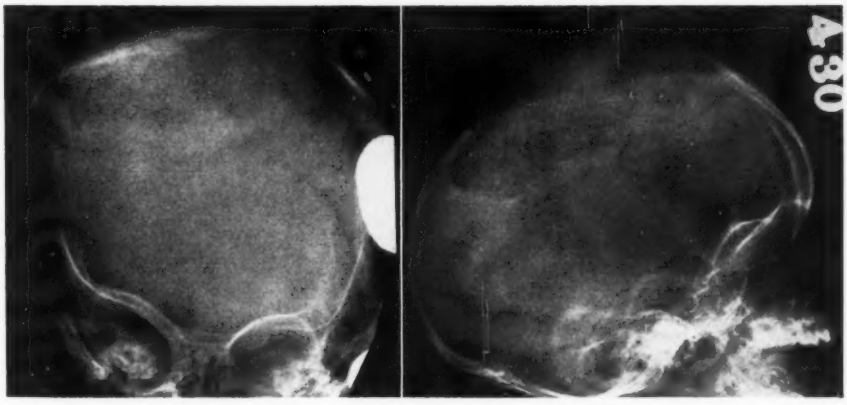


FIG. 13. Diagram of closure on S.

The progress of L. has been satisfactory. She began to walk at 16 months of age. Now at 28 months she talks well. An electroencephalogram on July 24, 1956 was reported as a normal tracing. The child weighs 32 pounds, the circumference of the head is 49 cm. and of the chest 50 cm. The skull defect measures 10 cm. in both the anteroposterior and transverse diameters. There is slight bulging of the scalp on the right side of the bony defect. It is not planned to attempt cranioplasty in this child for another 2 years.

The course of S. was complicated. At the initial dressing of her head, 2 days after the separation, a leak of cerebrospinal fluid was noted from the posterior part



FIGS. 14 and 15. Anteroposterior and lateral roentgenograms of skull of L. at 9½ months of age.

TABLE 3

Tabulation of operative procedures on S. after separation

1.	5-12-55	Skin grafting of exposed dura mater and cerebral protrusion—general anesthesia.
2.	5-21-55	Ventriculography—local anesthesia.
3.	5-26-55	Partial resection of cerebral hernia and suture of edge of dura mater to edge of scalp flap—general anesthesia.
4.	6- 2-55	Ventriculography—local anesthesia.
5.	7- 5-55	Skin grafting of cerebral hernia—general anesthesia.
6.	7-21-55	Ventriculography—local anesthesia.
7.	7-28-55	Ventriculocisternal shunt (Torkildsen)—general anesthesia.
8.	8- 2-55	Re-exploration of ventriculocisternal shunt—local anesthesia.
9.	8-16-55	Ventriculography—local anesthesia.
10.	8-25-55	Takedown of ventriculocisternal shunt and ventriculoperitoneal shunt—general anesthesia.
11.	9- 1-55	Re-exploration of ventriculoperitoneal shunt—general anesthesia.
12.	9-20-55	Takedown of ventriculoperitoneal shunt and ventriculoantral shunt—general anesthesia.
13.	9-27-55	Exploration of porencephalic cyst of right lateral ventricle and takedown of ventriculoantral shunt—local anesthesia.
14.	10- 4-55	Re-exploration of porencephalic cyst of right lateral ventricle—local anesthesia.
15.	10-29-55	Ventriculography—local anesthesia.

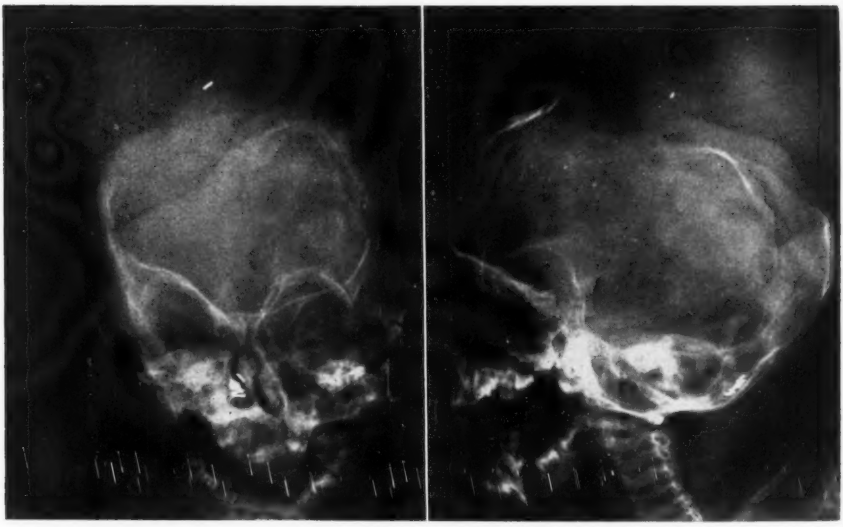
of the exposed dura mater on the right side. This persisted and by April 29 a cerebral hernia had begun to develop in the right posterior parietal region of the exposed dura mater. This area was not covered by the scalp flap in this child. The area of the herniation corresponded to the area of bulging that was noted later in the right side of the skull defect of L., but in that child this area was covered by the scalp flap.

The herniation of cerebral tissue in S. persisted and gradually increased in spite of repeated ventricular and spinal taps. Her course for the next 6 months is a complicated story of numerous operative procedures designed to correct the cerebral herniation. Initially the herniation increased in size in spite of the presence of continuous drainage of cerebrospinal fluid. When this finally ceased on July 1, 1955, further increase in the size of the protrusion was noted. A final skin graft over the protrusion on July 5, 1955 healed satisfactorily and from July 16, 1955 on there were no unepithelialized areas.

Table 3 is a chronological list of the surgical and diagnostic procedures carried out on S. after the separation of the twins on Apr. 21, 1955. None of the various shunts that were attempted were successful for more than a few days. Although there was some enlargement of the right ventricle the various ventriculograms failed to show significant generalized ventricular enlargement. It finally became apparent that attempts to drain or shunt cerebrospinal fluid from the ventricular system were futile and further that accumulation of the cerebrospinal fluid in the ventricule was not a significant factor in maintaining the protrusion.

S. was finally allowed to leave the hospital on Nov. 11, 1955. At that time her general condition was satisfactory. She weighed 12 pounds and 2 ounces and was 13½ months old. There was moderate torticollis, with the head rotated to the left. Because of the torticollis and the large cerebral protrusion the child could not hold her head up or sit up. There was moderate left spastic hemiparesis.

Figs. 16 and 17 are anteroposterior and lateral roentgenograms of the skull taken before dismissal. The size of the bony defect and the soft-tissue shadow of the protrusion can be seen. Since that time she has steadily improved. She now can sit up



FIGS. 16 and 17. Anteroposterior and lateral roentgenograms of S. at 13½ months of age.

alone and say a few words. There is still a marked left parietal cerebral hernia. At 28 months of age S. weighs 28 pounds, the circumference of her head exclusive of the hernia is 44 cm. and of the chest 50 cm. Fig. 18 is a photograph of the children at 2 years of age.

DISCUSSION

The successful surgical separation of craniopagus twins depends on the presence of separate brains with independent vascular circulations. Thorough preoperative study with adequate visualization of ventricles and subarachnoid spaces (pneumoencephalography or ventriculography) and the arterial and venous circulations (angiography) and the cooperation of neurologic and plastic surgeons and pediatricians and anesthesiologists are essential. If the initial scalp flaps are insufficient for closure, an adequate dural covering is important. In the case described the use of a fascial graft or of some plastic material or of tantalum mesh over the area of defective dura mater in the right parietal region of S. might have controlled the cerebral hernia and reduced the neurologic deficit in this child. It is felt, however, that the abnormal pre-separation ventriculogram in this child and the persistent evidence of obstruction of her cerebrospinal fluid pathways after separation indicate a congenital hydrocephalus which was largely the cause of our difficulty.

The problem of repair of the skull defect underlying the skin grafts remains to be solved in these children. A later report will deal with our attempts to repair these defects and correct the cerebral herniation of S.

FIG. 18. Photograph of children at two years of age. L. is on the left.

REFERENCES

1. BARBOSA, A. Tentativa cirúrgica em um caso de craniopagos. *Rev. brasil. Cir.*, 1949, *18:* 1047–1050.
2. BAUDOUIN, M. Un nouveau craniopage vivant: Emi-Lisa Stoll. *Sem. méd., Paris*, 1913, *33:* 553–555.
3. DABELOW, A. Ueber einen Fall von Spiegelbildlichkeit im Haarstrich eines Craniopagus. *Anat. Anz.*, 1926, *62:* 133–138.
4. GROSSMAN, H. J., SUGAR, O., GREELEY, P. W., and SADOVE, M. S. Surgical separation in craniopagus. *J. Amer. med. Ass.*, 1953, *153:* 201–207.
5. LÖWENBERG, K. Histological studies on the brain of a craniopagus. *Amer. J. Path.*, 1930, *6:* 469–476.
6. MÜNSTER, S. Cosmographiae universalis. Latin edition, 1559, Lib. 3, 625. French edition, 1575, *1:* Liv. 3, 1575. German edition, 1628, Book 5, 1059.
7. ROBERTSON, E. G. Craniopagus parietalis. Report of a case. *Arch. Neurol. Psychiat., Chicago*, 1953, *70:* 189–205.
8. ROGERS, J. B. Craniopagus monster. *J. Indiana med. Ass.*, 1936, *29:* 368.
9. SCHNEIDER, L. Beitrag zur Kasuistik der symmetrischen Doppelmissbildungen. *Zbl. Gynäk*, 1936, *60:* 1368–1372.
10. SONNENBURG, W. M. The blood vascular system in a parietal craniopagus. *J. Amer. med. Ass.*, 1919, *73:* 1345–1348.
11. SUGAR, O., GROSSMAN, H., GREELEY, P., and DESTRO, V. The Brodie craniopagus twins. *Trans. Amer. neurol. Ass.*, 1953, 198–199.
12. TRENKNER, W. Über einen Fall von Craniopagus parietalis. *Beitr. path. Anat.*, 1938, *101:* 223–236.
13. WARSCHAUER, O. Ein Beitrag zur Kenntniss des Craniopagus frontalis. *Arch. Gynaek.*, 1909, *89:* 498–507.

CASE REPORTS AND TECHNICAL NOTE

SPINAL CORD COMPRESSION BY AN INTRAMEDULLARY ANEURYSM

CASE REPORT AND REVIEW OF THE LITERATURE

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Congenital aneurysms of the vessels of the spinal cord have been reported only rarely. It is of interest, therefore, to present the case of a patient who manifested spinal cord symptoms caused by such a lesion. A review of the reported cases indicates that certain features in common may allow one to entertain a diagnosis.

CASE REPORT

I.P., a 41-year-old housewife, was admitted to the hospital on July 27, 1955, having enjoyed good health until February, 1954. After becoming pregnant, she had gradual onset of pain in the left forearm, the pain spreading upward across the chest anteriorly and down the right arm. After 3 days cervical traction was applied with relief of pain. On being mobilized she was slightly unsteady and ataxic. In July, 1954, she was delivered by Caesarean section. The pains in the arms reappeared, continued, and were accompanied by pains in both legs. These were aggravated by motions of the head and neck. Initiation of micturition became more difficult after the section. There was loss of bowel sensation and she had numbness of the buttocks. Two weeks prior to admission, her gait became rapidly weaker.

Examination. The patient was a well developed woman who could not stand unsupported. The cervical and thoracic spines were normal to gross inspection and palpation. Perception of pain was diminished bilaterally below T1, but was preserved in the left lower extremity. Loss of heat sensation, but preservation of perception of cold, was found below T4. Vibration sense was absent in both lower extremities. Postural sensation was absent in the left toes and ankle, but preserved in the right. There was marked weakness of the entire left lower extremity, while motor power was normal in the right. Motor power in both upper extremities was good, except for moderate weakness and atrophy of the intrinsic muscles of the hands, which were more pronounced on the left side. The fingers were thin and spindle-shaped. No fibrillations were evident. The deep tendon reflexes were active and equal. The toes were dorsiflexed on plantar stimulation.

The hemogram was normal and chemical studies of the blood revealed no abnormalities. Cerebrospinal fluid pressure was normal, with no evidence of obstruction. The fluid was clear and colorless, containing 1 lymphocyte per c.mm.; proteins were 50 mg. per 100 cc. The colloidal gold curve was normal; the Wassermann reaction was negative.

Roentgenograms of the chest were normal. Pantopaque myelography showed temporary arrest of the column at the level of the 7th cervical segment. The column flowed around this area, implying an intramedullary lesion.

The symptomatology was attributed to either a syringomyelic cyst or an intramedullary tumor.

Operation. On Aug. 2, 1955, a cervicodorsal laminectomy revealed a blue, discolored area beneath a tense dura mater, at the level of the 7th cervical segment. On incision and retraction of the dura mater, a rounded, blue, cystic intramedullary lesion of the left half of the spinal

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cord was uncovered (Fig. 1). The dorsal columns were spread thinly over this lesion. A needle, introduced into the lesion, recovered a chocolate-colored fluid. No fresh blood was encountered. The pia arachnoid, over the dome of the lesion, was incised longitudinally, and the remnants of the posterior columns were separated gently from it. The dome of the lesion was incised with the escape of more chocolate-colored fluid. With gentle dissection the well circumscribed lesion was separated from the medullary portion of the spinal cord, revealing two

FIG. 1. The spinal cord showed a blue, rounded mass within the left intramedullary portion beneath the posterior columns.

arterial vessels at its medial aspect. These were clipped. The main mass of the pea-sized structure was separated easily from the spinal tissue, leaving a capillary bed. During the course of dissection, and after delivery of the lesion, it appeared to have the gross characteristics of a thrombosed aneurysm. A needle was introduced into the cervical enlargement superiorly to eliminate the possibility of an associated syringomyelic cavity. None was encountered.

Postoperative Course. Paresthesias of both arms were troublesome for 5 days and then cleared gradually. By the 8th day the sensory changes had regressed almost completely on the left side, but only to T4 on the right. Motor power in the left extremity improved considerably and, with physiotherapy, the patient resumed walking. Sensibility and control of the bladder were regained. Three weeks after surgery the sensory changes, to pain and heat, were still detectable on the right below T6. There were none on the left. Vibration sense was absent in both toes and ankles. The postural sense was intact on the right and absent on the left. The toes were in plantar flexion. Gait had improved to the point where the patient could walk unsupported.

Pathological Report. The wall of the aneurysm (Fig. 2) was composed of collagenous tissue

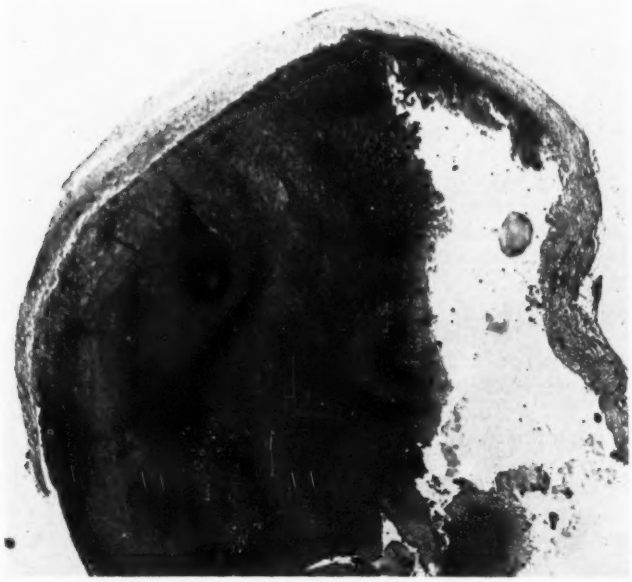

FIG. 2. The wall of the aneurysm is thin and composed of relatively acellular collagenous tissue. In the outer portion of the wall there is a moderate number of lymphocytes and plasma cells, accompanied by a few small engorged blood vessels. Within the wall a considerable amount of blood is present. This shows partial organization with strand-like areas of fibrin, within which are leucocytes.

and enclosed blood elements varying from a collection of fresh blood cells with leucocytic elements to smaller collections which were in various stages of organization and thrombosis. The walls of vessels with arterial components were identified at the base of the structure.

DISCUSSION

Arterial anomalies of the spinal cord produce their symptoms by external pressure and are usually associated with coarctation of the aorta or other types of congenital heart disease. Such lesions may show localized aneurysmal sacs similar to the congenital type ordinarily seen in the cerebral vessels.

Raymond and Cestan[4] reported a patient with a history of spastic paraplegia for 20 years caused by a cirsoid aneurysm of the anterior and posterior spinal vessels. The aneurysm extended from C2 to C7 and was extramedullary and intramedullary in position. The posterior aspects showed aneurysmal dilatations but the lesion did not appear to be congenital in type. Hebold[2] found multiple miliary aneurysms in the extramedullary and intramedullary vessels of the spinal cord of a young girl. Wyburn-Mason[7] discovered 5 instances of localized aneurysm associated with arterial anomalies.

Symptoms in Sargent's[6] case began with weakness and wasting of the right arm and, after gradually increasing compression over 2 years, ended with a complete transverse lesion at the T1 segment. A local aneurysm of the anterior spinal artery, which compressed and destroyed the cord at C7 segment, was found at autopsy.

The clinical course in Wyburn-Mason's[7] Case 31 was initiated by spinal sub-

arachnoid hemorrhage, followed by temporary weakness of one leg and gradually increasing spinal compression over a 2-year period. Postmortem examination divulged a localized aneurysm arising from a tortuous and dilated anterior spinal artery, causing compression and destruction of the cord. In his Case 32, progressive weakness of both hands with increasing numbness of the upper part of the chest characterized the clinical course. Autopsy showed compression of the cord by an aneurysm formed from the anterior spinal artery at the level of the T3 segment. In each of the reported cases the lesion was associated with coarctation of the aorta.

Lorenz'[3] patient had weakness of the left arm followed by sudden motor and sensory paralysis. Postmortem examination disclosed a cherry-sized angioma at the 7th cervical segment with hemorrhage into the subarachnoid space and substance of the cord from C1 to C7.

Robertson[5] reported a girl of 16 whose initial symptoms were pains in the back and the right arm. Subsequently there developed paralysis of the right arm and leg, followed by paralysis of the left side. There were respiratory difficulties. Postmortem examination revealed a racemose arterial aneurysm with a localized laminated blood clot which had reduced the lower three cervical segments to a crescentic remnant.

Babonneix and Widiez[1] reported a man of 56 with a clinical picture of combined sclerosis and syphilis. An illustration of the postmortem specimen demonstrated graphically an aneurysm of the lower cervical portion of the anterior spinal artery, to which the symptomatology was ascribed.

In our case there was no clinical evidence of coarctation of the aorta or other detectable congenital cardiac lesion. Symptoms began with severe neuritic pains in the upper extremities accompanied by atrophy of the intrinsic muscles of the hands. Dissociation of sensory changes as well as the atrophy led to a diagnosis of syringomyelia. Resection of a well circumscribed, organized, vascular, intramedullary structure along the course of the arterial channels allowed a diagnosis of aneurysm at the time of surgery. Unfortunately the vessel of origin of the aneurysm could not be established. The residuals, at the time of publication, consist of weakness and ataxia caused by loss of postural and vibration sensation in the left lower extremity.

The diagnosis of congenital aneurysm of the arteries of the spinal cord should be entertained when the syndrome of cord compression is accompanied by clinical evidence of coarctation of the aorta or other cardiac anomalies. In some cases the clinical course is initiated by the signs of subarachnoid hemorrhage with localization in the spinal cord. A point of considerable diagnostic significance is the frequent localization of the lesion to the cervicothoracic junction. When there are pain and atrophy in the upper limbs, with the gradual development of a Brown-Séquard or compression syndrome, the diagnosis of aneurysm of the anterior spinal artery may be suspected.

No one feature in the spinal fluid is diagnostic of the condition, but the finding of fresh blood and the demonstration of an intramedullary mass by myelography at the cervicothoracic junction strengthen the diagnosis. We have seen no reports of vertebral angiography, aortography or intraosseous venography having been employed in the attempt to visualize the lesion.

SUMMARY

1. A case is reported of a spinal cord lesion caused by an arterial aneurysm.

2. A brief review is made of the clinical and pathological findings in previously reported cases.

3. The criteria are discussed for diagnosis of aneurysm of the spinal arteries.

REFERENCES

1. Babonneix, L., and Widiez, A. Sclérose combinée; lésions diffuses et inflammatoires du névraxe. Anévrysme de l'artère spinale antérieure. Syphilis probable. *Rev. neurol.*, 1930, *1:* 1214–1217.
2. Hebold, O. Aneurysmen der kleinsten Rückenmarksgefässe. *Arch. Psychiat. Nervenkr.*, 1885, *16:* 813–823.
3. Lorenz, F. O. Cavernöses Angiom des Rückenmarks. Tötliche Blutung. (Thesis) *Jena:* 1901.
4. Raymond, and Cestan, R. Un cas d'anévrisme cirsoide probable de la moelle cervicale. *Rev. neurol.*, 1904, *12:* 457–463.
5. Robertson, E. G. A case of arterial angioma of the spinal cord. *Med. J. Aust.*, 1938, *2:* 384–385.
6. Sargent, P. Haemangeioma of the pia mater causing compression paraplegia. *Brain*, 1925, *48:* 259–267.
7. Wyburn-Mason, R. The vascular abnormalities and tumours of the spinal cord and its membranes. *London: H. Kimpton*, 1943, vii, 196 pp. (see p. 37).

CHOROID PLEXUS PAPILLOMA CAUSING SPONTANEOUS SUBARACHNOID HEMORRHAGE

REPORT OF CASE AND REVIEW OF LITERATURE

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Among the non-aneurysmal causes of bleeding into the subarachnoid spaces are vascular malformations and vascular tumors. Of the latter, papillomas of the choroid plexus have only recently been implicated as a cause of blood appearing in the cerebrospinal fluid. The following case represents such an unusual lesion which was recognized and treated during life, though terminating fatally because of complications.

REPORT OF CASE

J.G., a 15-year-old school girl, was admitted to the Ohio State University Hospital on June 28, 1955. She had been well until 3 days prior to this when there developed sudden severe frontal headache associated with vomiting, slight nuchal rigidity, mental fogginess and drowsiness. Examination disclosed bloody (cherry red) cerebrospinal fluid under 280 mm. of pressure. By the morning of admission, she was stuporous with paroxysms of severe headache and vomiting.

Examination. There was bilateral early papilledema with unequal pupils, the left being slightly larger than the right. A positive left Babinski sign was present. On lumbar puncture the cerebrospinal fluid was found to be under 300 mm. of pressure and contained 29,000 red blood cells and 65 white blood cells per c.mm., with a total protein content of 66 mg. per cent. Bilateral carotid angiograms revealed a questionable vascular anomaly in the right posterior superior temporal area.

Course. Bilateral papilledema increased rapidly to 3 D. with associated retinal hemorrhages. On June 30, 1955, a vertebral angiogram (Fig. 1) disclosed the vascular anomaly in the right posterior superior area. On July 2, her condition worsened with increase in papilledema, a slow pulse and increasingly bloody cerebrospinal fluid containing a total protein content of 1,280 mg. per cent. Bilateral posterior ventriculostomy disclosed blood clots in the right lateral ventricle. Ventriculograms revealed moderately dilated lateral and third ventricles without displacement and a filling defect in the right lateral ventricle at the trigonal area, considered to be a mass lesion—either the remains of a blood clot or a neoplasm.

Operation. On July 4, 1955, through a parieto-occipital transcortical approach, a tumor, about 3×2×2 cm. in size, was found within the trigonal area of the right lateral ventricle, extending anteriorly proximal to the foramen of Monro and for a short distance into the temporal horn. Its arterial supply was multiple, with at least four vessels from the floor and medial wall of the body of the lateral ventricle and others from the floor and wall of the temporal horn. One large vein was seen to drain the blood, probably into the internal cerebral veins. As these vessels were clipped the size of the tumor decreased most remarkably so that upon its removal it was about one-third its original mass.

Course. Her immediate postoperative course was unsatisfactory the next 2 days. Re-

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exploration was necessary on July 18 (14th postoperative day) and disclosed severe cerebral edema and blockage of the foramen of Monro with blood clot. A Torkildsen ventriculocisternostomy was established as a precautionary measure. In the following days there developed troublesome accumulations of subgaleal fluid, projectile vomiting and very severe headaches. On July 26 (22nd postoperative day), re-exploration disclosed an epidural blood clot of moderate size with considerable cerebrospinal fluid locked in this area, having come out alongside of the Torkildsen tube, and the latter was blocked with blood clots. She appeared to improve for about 36 hours, then left hemiplegia developed, and she lapsed into coma and died on July 31 (27th postoperative day).

Postmortem Examination. The brain was edematous with severe swelling in the posterior half of the right cerebral hemisphere. Infarction of the brain in the area supplied by the

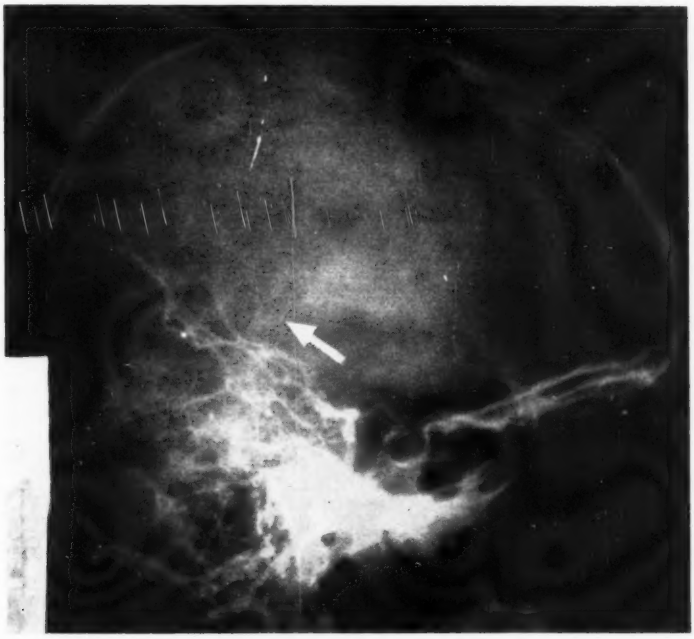

FIG. 1. Vertebral angiogram (lateral view) disclosing "vascular fringe" (arrow), which is believed to outline part of the papilloma in the right lateral ventricle.

right posterior cerebral artery was evident. Severe uncal herniation through the tentorial incisura with consequent pressure against the posterior cerebral artery was considered to be the cause of the encephalomalacia. Retrograde thrombosis from the extensive vascular supply to the site of the tumor could not be ruled out as a contributory or a primary cause.

Histopathology of Tumor. The tumor (Fig. 2) presented the characteristic appearance of a papilloma of the choroid plexus. The villi-like structures were covered with cuboidal cells whose cytoplasm was granular and contained minute vacuoles and a central nucleus. The central portion of the villi was made up of loose and somewhat scanty connective tissue, but was rich in blood vessels. The absence of glial fibers and blepharoplasts differentiated it from a papillary ependymona.

Comment. The unfortunate loss of the patient in this case does not deter from its

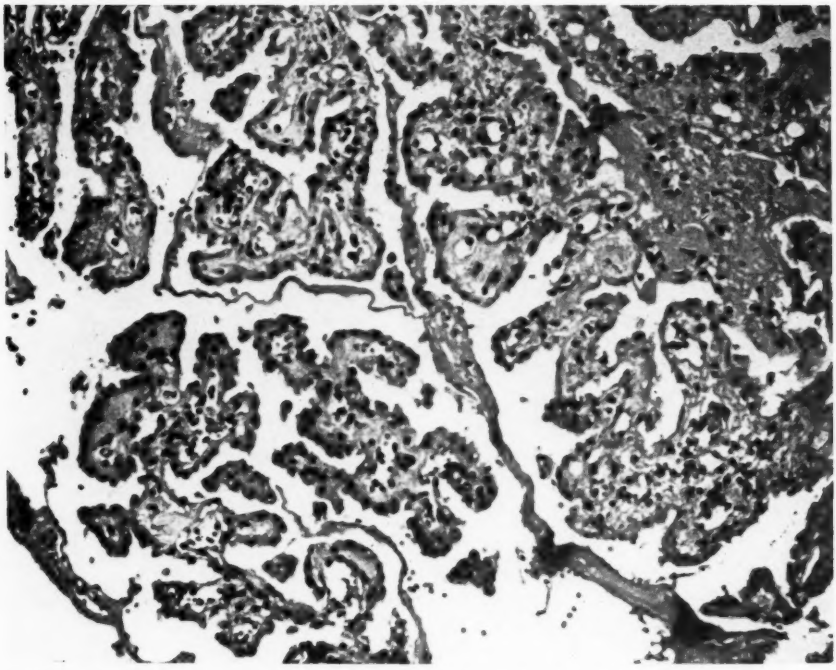

FIG. 2. Photomicrograph of papilloma of choroid plexus showing its characteristic papillary arrangement and loose connective tissue containing blood vessels and covered with rather uniform cuboidal cells. Hematoxylin and eosin, ×200.

clinical significance and interest. The presence of blood in the right lateral ventricle with angiographic evidence of a probable vascular anomaly in the ventricle and a mass defect in the ventricle completed the picture that is diagnostic of the possibility of a papilloma or other mass in the ventricle.

Criticism of the manner in which this case was handled surgically may well be in order. Difficulties were encountered in controlling the bleeding from the tumor bed at the first operation and undoubtedly this was a primary factor in subsequent complications since blood clot was present in the ventricle at the second operation. In retrospect it may be apparent that certain changes in the surgical approach might have proved efficacious, such as (1) a more radical removal of brain tissue in approaching the ventricle (removal of a core of brain instead of a spreading cortical incision); (2) a little lower approach on the hemisphere (incision over the trigonal area instead of through the wound of the occipitoparietal ventriculostomy); and (3) it might have been far better to have left the dura mater open and the bone flap loose or out entirely, thus allowing for cerebral swelling.

DISCUSSION

While it is true that in most cases of subarachnoid hemorrhage the cause is leaking intracranial aneurysm, it is likewise apparent that in from 20 to 40 per cent

of the cases complete cerebral angiography fails to reveal such a lesion.* In two previous reports, one of us (KHA)[1,4] has emphasized that intraspinal and intracranial neoplasm may give rise to subarachnoid hemorrhage. Of 41 reported cases of intracranial tumor with associated subarachnoid hemorrhage about 50 per cent were primary gliomas of the brain, 25 per cent were pituitary tumors and nearly 15 per cent were intracranial vascular tumors.

Bleeding from a papilloma of the choroid plexus, to our knowledge, was first described by Graves and Fliess[5] and subsequently by Friedman and Solomon;[3] however, in these cases the tumor was found at necropsy. Ernsting[2] appears to have been the first to report a case in which the diagnosis was made clinically and operation was performed. The patient was a 26-year-old male who had had at least one previous (by 3 years) episode of intraventricular bleeding. The second attack was sudden in onset and characterized by slight mental confusion, slight nominal aphasia, a right upper quadrantic homonymous hemianopsia and slight lower facial weakness. Left carotid angiography was done and an "intracerebral vascular lesion, measuring 3 cm. in its antero-posterior diameter and 1.5 cm. in its vertical diameter, was demonstrated in the left posterior temporal region. It was supplied by a vessel arising from the internal carotid artery just proximal to its bifurcation, presumably the left anterior choroidal artery." Pneumoencephalography confirmed the presence of a mass occupying the greater part of the trigonal region of the left lateral ventricle and it "appeared to change its position with alterations in the posture of the head." At the first operation via the temporal lobe, only a biopsy was done, but on the second approach to the tumor, a more adequate temporal exposure allowed complete extirpation with subsequent uneventful recovery.

The diagnosis of an intraventricular mass was made, in the case of Ernsting[2] and in our case, through both angiography and pneumoencephalography. As more of these lesions are surely to be reported, however, it may well be that these minor vascular changes as shown by angiography in the trigonal region of the lateral ventricle or the temporal horn itself will prove of diagnostic significance and thus delay in their diagnosis and surgical extirpation will be avoided.

SUMMARY

It has been emphasized that severe subarachnoid hemorrhage may occur from papillomas of the choroid plexus as well as other neoplasms and from vascular malformations. In this report a papilloma of the choroid plexus in the right lateral ventricle was shown to be the source of severe intraventricular hemorrhage. Cerebral angiography and ventriculography were helpful in establishing the presence of the tumor which was surgically removed. This appears to be the second such case to be diagnosed clinically. Two others from autopsy material have been described previously.

It is suggested that in those cases in which angiography fails to disclose the etiology of subarachnoid hemorrhage, further studies—closer scrutiny of technically better angiograms together with ventriculography—may occasionally prove fruitful

* These figures are only an approximation gathered from various series in the literature. Hamby[6] has reported, "In a series of patients surviving subarachnoid hemorrhage, a smaller percentage of aneurysms (41 per cent) was disclosed by angiography than in a series studied at autopsy (93.6 per cent)." The accuracy of any such figures is questionable since it is the consensus that in those cases in which there is no aneurysm, subarachnoid hemorrhage is less likely to be fatal and to recur, hence less likely to appear in autopsy material for comparison.

in establishing the source of the hemorrhage in such a lesion as choroidal papilloma or other tumor.

REFERENCES

1. Abbott, K. H. Subarachnoid hemorrhage from an ependymoma arising in the filum terminale. Report of case. *Bull. Los Angeles neurol. Soc.*, 1939, *4:* 127–132.
2. Ernsting, J. Choroid plexus papilloma causing spontaneous subarachnoid haemorrhage. *J. Neurol. Neurosurg. Psychiat.*, 1955, n.s. *18:* 134–136.
3. Friedman, J. J., and Solomon, C. I. Tumors of the choroid plexus in childhood. *Amer. J. Dis. Child.*, 1936, *52:* 114–127.
4. Glass, B., and Abbott, K. H. Subarachnoid hemorrhage consequent to intracranial tumors. Review of literature and report of seven cases. *Arch. Neurol. Psychiat., Chicago*, 1955, *73:* 369–379.
5. Graves, G. W., and Fliess, M. M. Neoplasm of the choroid plexus. Report of a case with review of the literature. *Amer. J. Dis. Child.*, 1934, *47:* 97–103.
6. Hamby, W. B. The aneurysmal origin of nonfatal subarachnoid hemorrhage. An angiographic survey of 53 cases. *J. Neurosurg.*, 1953, *10:* 35–37.

DIASTASIS OF THE OPTIC NERVE

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Anomalies of the optic nerve are not rare. The optic disc, the intraocular portion of the nerve, has many variations in structure and many forms that are termed anomalous. They are readily observed with the ophthalmoscope. The other three portions (intraorbital, intraosseous and intracranial) perhaps are just as frequently anomalous, but are seldom observed.

Duke-Elder,[1] under the term "division of the optic nerve," introduces a brief discussion of this condition as follows, "As a rare anomaly the optic nerve may be divided into two strands, of which the smaller accessory bundle contains the uncrossed fibres. . . ."

No attempt has been made at an exhaustive survey of the literature, but the only similar anomaly described with an illustration is reproduced herewith (Fig. 1).[2]

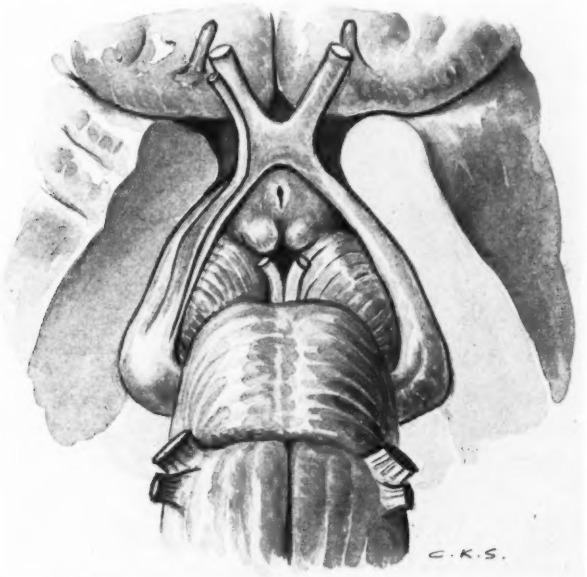

FIG. 1. Splitting of the right optic nerve and right optic tract described by Ganser[2] in 1882. It was felt that the uncrossed fibers composed the anomalous bundle.

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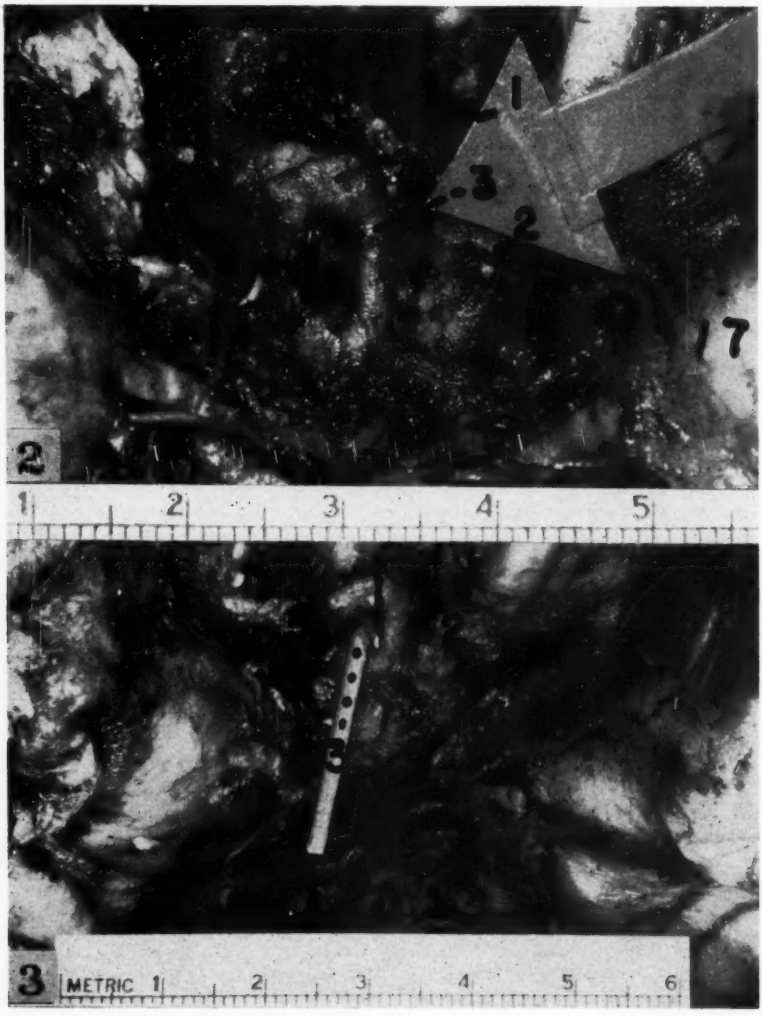

FIGS. 2 and 3. Photographs of the diastasis (1) and anomalous vessel (3). The diastasis measures 5 mm. in length.

Key for Figs. 2, 3 and 4: 1. Diastasis of right optic nerve. 2. Left optic nerve. 3. Auxiliary right anterior cerebral artery involved in diastasis. 4. Aneurysm of anterior communicating artery. 5. Right anterior cerebral artery. 6. Trifurcation of right internal carotid artery. 7. Right middle cerebral artery. 8. Right posterior communicating artery. 9. Right posterior cerebral artery. 10. Right superior cerebellar artery. 11. Basilar artery. 12. Vertebral arteries. 13. Left superior cerebellar artery. 14. Left posterior cerebral artery. 15. Left posterior communicating artery. 16. Left middle cerebral artery. 17. Left internal carotid artery. 18. Left anterior cerebral artery.

Personal communication with authorities in the field of human embryology and anatomy has failed to reveal any further information as to occurrence of or an explanation for the anomaly.[3,4]

The specimen we wish to record was discovered at autopsy.

CASE REPORT

Cleveland City Hospital No. 324779. H.D., a 43-year-old negro male, suddenly became unconscious the day before admission on Oct. 22, 1951. He regained consciousness in 30 minutes and complained of a stiff neck.

Examination. Blood pressure was 135/70, temperature 36.5°C., pulse rate 52, and respiratory rate 16. The patient was lethargic and confused. The pupils were equal but did not react well to light. There was papilledema of the left optic disc. The neck was stiff. Repeated lumbar punctures yielded bloody spinal fluid with cerebrospinal fluid pressures varying from 280 to 340 mm. of water.

Past history revealed syphilis in 1944, but the last serology was negative. Neither visual acuity nor history of visual difficulty was recorded. Four days after admission the patient had a sudden respiratory arrest.

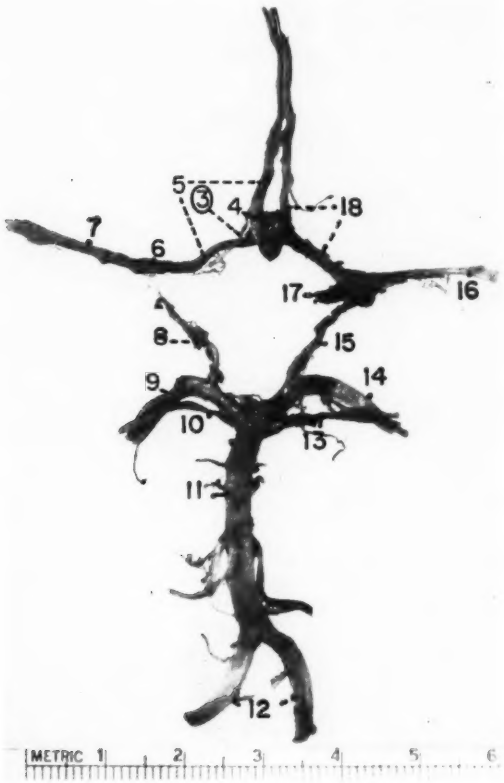

FIG. 4. Photograph of the circle of Willis (see Figs. 2 and 3 for explanation of numerals).

Postmortem examination of the brain revealed a large collection of fresh blood clots in the middle cranial fossa and over the base of the brain. At the base of the brain the right internal carotid artery had an anomaly consisting of an abnormal branch arising at a point where the right internal carotid lay on the external surface of the right optic nerve. This abnormal branch went through the right optic nerve, producing a diastasis in this nerve, the diastasis measuring approximately 5 mm. in length (Figs. 2 and 3). This abnormal branch from the internal carotid ended near the anterior pole of the right frontal lobe at the right anterior cerebral artery distal to the anterior communicating artery. The true anterior cerebral arteries of each side were normal in position and course. At the site of the anterior communicating artery an aneurysmal sac was present, measuring 1.5 cm. in diameter in the fresh state (Fig. 2.)

Comment. It appears that the anomalous blood vessel was responsible for the splitting of the right optic nerve during the centripetal growth of the optic nerve. The gross specimen was destroyed before the relationship of the crossed and uncrossed fibers could be studied.

SUMMARY

A case report of diastasis of the right optic nerve caused by an abnormal branch of the right internal carotid artery is presented.

The photographs of the gross pathology in this case are gratefully acknowledged to the Pathology Department, Cleveland City Hospital.

REFERENCES

1. Duke-Elder, W. S. Text-book of ophthalmology. *St. Louis: C. V. Mosby Co.*, 1938, *2:* 1346.
2. Ganser, S. Ueber die periphere und centrale Anordnung der Sehnervenfasern und über das Corpus bigeminum anterius. *Arch. Psychiat. Nervenkr.*, 1882, *13:* 341–381.
3. Hoerr, N. Personal communication.
4. Padget, D. Personal communication.

ARTERIAL GRAFT AND ENDARTERECTOMY IN RECONSTITUTION OF THE COMMON CAROTID ARTERY TWO YEARS FOLLOWING LIGATION

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Developments in the surgery of blood vessels and increasing interest in factors underlying cerebral ischemia have led to attempts to restore the internal carotid circulation after occlusion by obliterative disease.[3,4,5]

This is a report of reconstitution of the common carotid artery by endarterectomy and homograft, 2 years after its surgical ligation.

CASE REPORT

A 44-year-old man was admitted to the hospital on May 1, 1956, because of difficulty in the use of his right arm and leg for 1 week.

Two years previously, he had been treated in a hospital elsewhere for subarachnoid hemorrhage, characterized by malaise for 1 day, followed by severe headache, stiff neck, vomiting, and drowsiness, but without focal neurological disturbance. Initially, the spinal fluid was bloody; later it was deeply orange in color. An angiogram made on the 19th day showed no abnormality. On the 29th day, the left common carotid artery was tied 3 cm. proximal to its bifurcation. Recovery was complete except for a minor impairment of memory, and the patient returned to his work as an attendant in a filling station.

Approximately 2 years after the ligation of the common carotid artery, and 1 week before his present admission, movements of his right arm and leg became awkward. He dropped objects held in the right hand, and was unable to use this hand in eating. Weakness of the right leg was observed, particularly when he attempted to arise from the sitting position. He was forced to give up his work.

By the time of his admission to the hospital, most of these symptoms had disappeared, and on neurological examination only weakness of dorsiflexion of the right foot was found. The operative site in his neck was well healed and a definite, though diminished, carotid pulse could be felt above this point.

Angiography, May 9, 1956. On the right side, normal filling and distribution of the internal carotid, anterior and middle cerebral vessels were demonstrated. On the left, the needle was inserted into the vessel above the cervical scar, and although the external carotid artery was well visualized, the internal carotid artery filled only to the base of the skull (Fig. 1).

The episode of right hemiparesis was interpreted to be the result of cerebral vascular insufficiency in the distribution of the left carotid, based on the surgical ligation performed 2 years previously. Because no aneurysm or other vascular lesion was evident in the left carotid angiogram made before ligation of the common carotid, it was decided to try to re-establish blood flow through the left carotid system.

Operation, May 24, 1956. The carotid bifurcation in the left side of the neck was exposed. Although somewhat diminished in caliber, both the external and the internal carotid arteries were patent and pulsating. With occlusion of the external carotid artery, a good back flow through the internal carotid artery was demonstrated. The entire length of the common carotid artery from its bifurcation to the arch of the aorta was exposed, necessitating an incision down the neck and over the clavicle, resection of the medial portion of the clavicle, and splitting of the sternum to its midportion with a Lebsche knife (Fig. 2). Obliterating the lu-

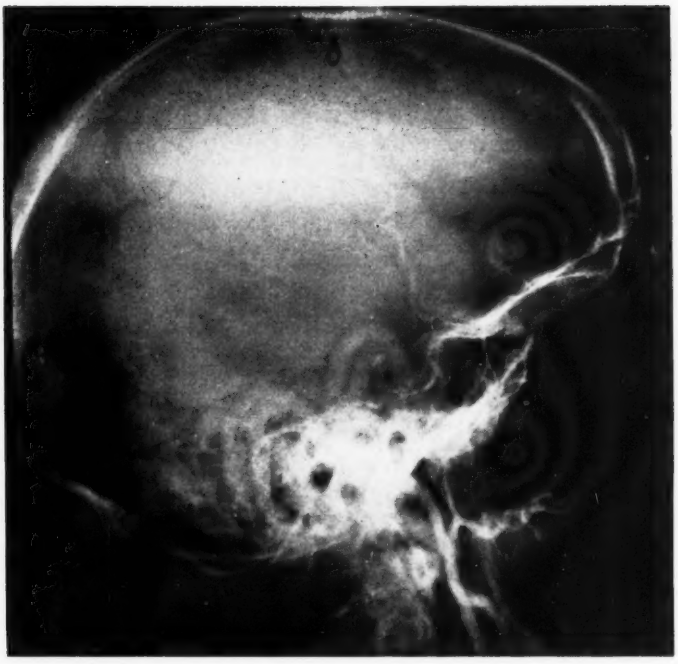

FIG. 1. The cervical part of the internal carotid artery is filled, but the column of dye cannot be followed beyond the base of the skull.

men of the common carotid artery from the aorta to the bifurcation was a firm organized thrombus, and surrounding the upper segment of the artery and its bifurcation was a dense scar, marking the site of the previous ligation. It was felt that blood flow could be re-established between the common carotid artery below the ligation and the internal carotid artery above. Therefore, the thrombus was removed by the use of a circular intraluminal stripper (Fig. 3) as employed for endarterectomy in the lower extremities.[2] Despite the fact that the thrombus had been in place for 2 years, it was relatively easy to insert the stripper between it and the normal intima and to dissect the clot from the wall of the vessel. Slight resistance was felt as the stripper entered the aortic arch, and immediately thereafter the loosened thrombus (measuring 17 cm. in length) was blown out the open end of the carotid artery by a gush of blood.

Because of the injury of the vessel at the point of its previous ligation, it was necessary to excise 5 cm. of the common carotid artery and a short cuff on each side above its bifurcation. A lyophilized iliac homograft was used to bridge the lower segment of the common carotid artery to the internal carotid artery, the external carotid artery being sacrificed (Fig. 3). Upon release of the occluding clamps, a bounding arterial pulse occurred in the carotid artery, and its caliber was greatly increased.

Apart from a paralysis of the mandibular branch of the left facial nerve, there were no postoperative difficulties.

Postoperative Angiography, July 9, 1956, 7 weeks after operation. A left common carotid arteriogram revealed continued patency of the left common carotid artery and a normal pattern of cerebral vessels (Fig. 4).

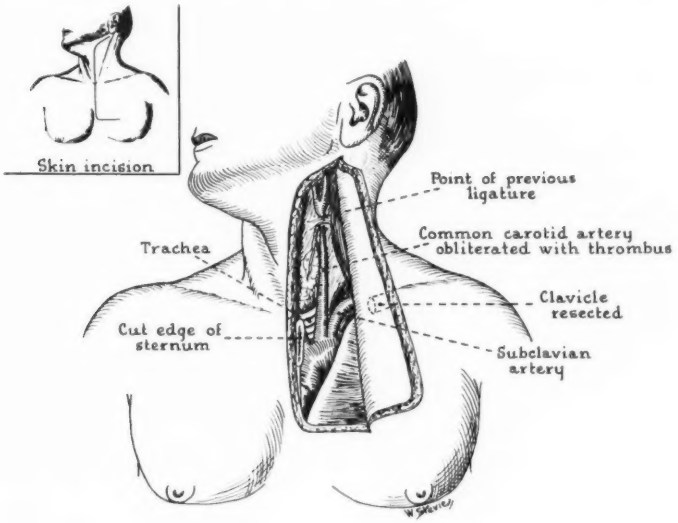

FIG. 2. Incision and operative exposure.

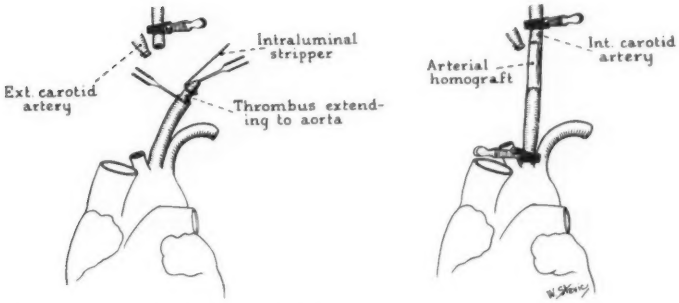

FIG. 3. Operative steps, showing removal of thrombus from the proximal segment of the common carotid artery and the vessel after reconstruction and prior to removal of occluding clamps.

DISCUSSION

Although the precise circumstances that characterize this case must be extremely unusual, questions of broader importance and more general interest arise.

The first deals with interpretation of the carotid angiogram made distal to the ligature. It will be recalled that the internal carotid artery contained no contrast medium beyond the base of the skull. Before exploration, it was thought that this might be caused either by a separate occlusion high in the internal carotid artery, beyond the reach of presently available surgical procedures, or by blood flow in-

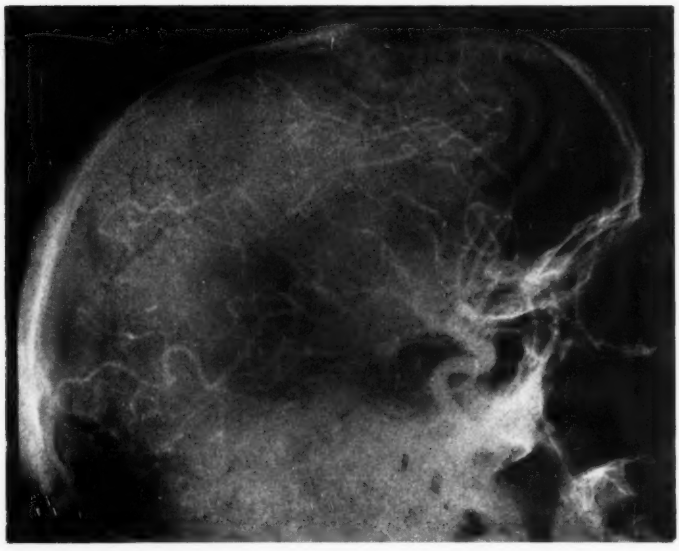

FIG. 4. Postoperative angiogram, showing the filling of the cerebral vessels on the left.

sufficient to sweep the dye distally in this artery. The latter was proved to be the case at operation and by subsequent arteriograms.

The technique of clearing the obliterated common carotid artery 2 years after ligature and thrombosis deserves comment. Clearly, the area of ligation required excision and replacement by a graft. The rest of the vessel was normal except for the occluding thrombus, which was easily dislodged by an intraluminal stripper. Although proximal stripping of this artery might be performed blindly, as is practised in the thigh, a tear of the unexposed artery within the thorax would be disastrous. Therefore, surgical procedures on the carotid artery in this area must be done under direct vision, regardless of the local problems of exposure. Methods for exposure of the major vessels within the superior mediastinum have been developed by Elkin and Shumacker.[6]

Cerebral ischemia is the most feared result of carotid ligation. As usually described, early and late forms are separated,[7] the former occurring immediately or within a few hours and the latter appearing after a delay of 24, 48, or even 72 hours. Thus, the cerebral circulatory failure following carotid ligation ordinarily has its onset within several days of the time when the artery is closed. This contrasts with the now widely recognized fact that, in spontaneously occurring obstruction, symptoms of ischemia often commence long after the occlusion is established. Why, following surgical occlusion, these long delayed difficulties are not prominent is difficult to understand. Perhaps insufficient time has elapsed for the subjects of larger published studies to have come forward in compelling numbers with this complication, or perhaps the relative rapidity of even gradual surgical occlusion has separated off at an early date most of those in whom collateral circulation is marginal—those who might under different circumstances have shown long delayed evidences of cerebral vascular insufficiency.

Finally, when surgical closure of a carotid artery seems necessary or desirable, consideration should be given to the fact that internal carotid ligation is irrevocable after a short interval, while common carotid closure is not. The possibility of re-establishing circulation at a later time might be weighed in selecting the site, especially since the available evidence fails to support clear superiority in terms of effectiveness and safety of either one over the other.[1]

SUMMARY

A 44-year-old man began to exhibit symptoms of carotid insufficiency 2 years after the left common carotid artery was ligated. Blood flow was re-established after endarterectomy and arterial graft.

REFERENCES

1. Brackett, C. E., Jr. The complications of carotid artery ligation in the neck. *J. Neurosurg.*, 1953, *10:* 91–106.
2. Cannon, J. A., and Barker, W. F. Successful management of obstructive femoral arteriosclerosis by endarterectomy. Experience with a semiclosed technique in selected cases. *Surgery*, 1955, *38:* 48–59.
3. Cooley, D. A., Al-Naaman, Y. D., and Carton, C. A. Surgical treatment of arteriosclerotic occlusion of common carotid artery. *J. Neurosurg.*, 1956, *13:* 500–506.
4. Denman, F. R., Ehni, G., and Duty, W. S. Insidious thrombotic occlusion of cervical carotid arteries, treated by arterial graft. A case report. *Surgery*, 1955, *38:* 569–577.
5. Eastcott, H. H. G., Pickering, G. W., and Rob, C. G. Reconstruction of internal carotid artery in a patient with intermittent attacks of hemiplegia. *Lancet*, 1954, *267:* 994–996.
6. Elkin, D. C., and Shumacker, H. B., Jr. Arterial aneurysms and arteriovenous fistulas. Technical considerations in their surgical management, with special reference to exposure of the blood vessels. In: *Surgery in World War II. Vascular surgery.* D. C. Elkin, and M. E. DeBakey, Ed. Washington, D. C.: Office of the Surgeon General, Dept. of the Army, 1955, ix, 465 pp. (see pp. 225–263).
7. Hamby, W. B. Intracranial aneurysms. *Springfield, Ill.: Charles C Thomas*, 1952, xxi, 564 pp. (see pp. 62–72).

PAGET'S DISEASE OF THE AXIS CAUSING QUADRIPLEGIA

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Paget's disease of the axis is an uncommon condition. Only one case, reported by Whalley,[7] was found in a review of the medical literature. In Whalley's case the atlas and the axis were both affected as well as the skull and there was an atlanto-occipital dislocation with paraplegia. In the case reported below the axis alone was affected and caused quadriplegia.

CASE REPORT

M. NS. 2635. A male aged 60 was admitted to the General Hospital on Oct. 10, 1953. For 15 years he had suffered pains all over the body, and during the last 5 years stiffness of the neck had developed with pain in the back of the neck. Three years prior to admission he noted weakness of the right lower extremity. Gradually this weakness involved the left lower limb also. During the last 6 months weakness spread to both upper limbs.

Examination. The patient was an ill-nourished old man. He was not able to walk or use his hands properly for eating or dressing. Intelligence and memory were good. The optic discs and all cranial nerves were normal. The size of the head was normal. The neck was stiff and the patient was not able to move the head freely in all directions. There was fullness in the upper part of the back of the neck. There was no tenderness anywhere on the spine. Muscle power in all four limbs was diminished and the tone was spastic. There was loss of appreciation for all modalities of sensation up to the level of the nipple. Deep reflexes in all extremities were exaggerated. Abdominal and cremasteric reflexes were lost. Plantar responses were extensor. The patient had retention of urine with overflow.

A diagnosis of cord compression at the cervical level was made. Roentgenograms of the cervical spine showed sclerosis of the 2nd cervical vertebra (Figs. 1 and 2).

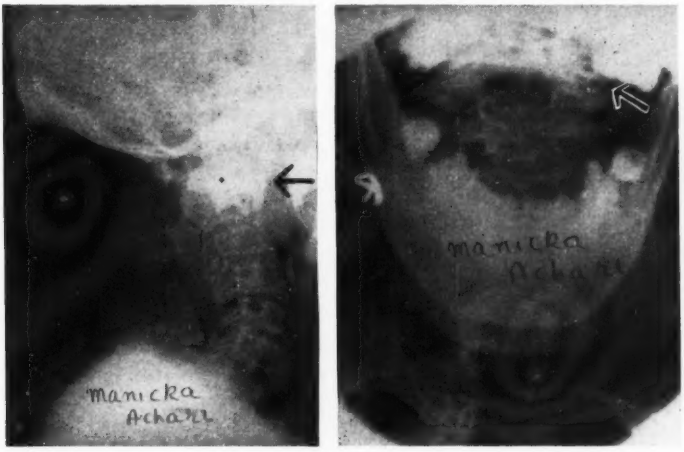
The following diagnoses were entertained: (a) localized fluorosis, (b) localized spondylitis of the cervical vertebrae, and (c) secondary malignant bone deposit.

The blood Wassermann reaction was normal.

Lumbar puncture was done. There was a complete block on Queckenstedt's test. The cerebrospinal fluid proteins were 80 mg. per cent; there were no cells; and Wassermann reaction was negative. A myelogram done after cisternal puncture showed a block at the level of the 2nd cervical vertebra.

Operation. Anaesthesia was induced by intravenous Pentothal. An endotracheal catheter was introduced and anaesthesia was maintained with gas and oxygen. An incision was made in the mid line in the back of the neck. After separation of the muscles, the ligamentum nuchae was split and the spine of atlas and the axis were visualized. At this stage the patient was not breathing properly and respiration was maintained artificially by compressing the bag. The laminae of the axis were thickened, soft and very vascular. These were removed piecemeal and the cervical dura mater was exposed. The spinal cord was found to be kinked at the level of the body of the axis. The arch of the atlas was also removed to provide a good decompression and the wound was closed.

Course. At the end of the operation the patient still had difficulty in respiration, which had to be assisted. An hour later the patient regained consciousness. But still he could not breathe by himself. He was kept in this condition for 72 hours by artificial respiration. The trachea was kept clear by repeated aspiration. He was fed by a Ryles tube. On the 4th day the patient had cardiac failure and expired.



FIGS. 1 and 2. Roentgenograms of cervical spine, showing increased density of the axis. (*Left*) Lateral view. (*Right*) Anteroposterior view (patient's mouth open).

Autopsy. The axis was enormously thickened, and there was a well marked hump at the level of the axis. The cord showed evidence of intramedullary haemorrhages.

Microscopical Examination. Irregular segments of the lamellae of bone are separated by short irregularly toothed cement lines. These appear fitted into one another and form an irregular mosaic. There is no attempt to form Haversian systems. The marrow between the bony trabeculae is fibro-fatty and in a few places consists of myeloid cells. There are also small isolated bits of deeply staining irregular trabeculae in the midst of the marrow. Of the lamellar pieces forming the mosaic a few stain differently from the others with haematoxylin, from light bluish- to purplish-red. There are also areas of reconstructed bone staining deep purplish-brown, irregular in their outline and extent, which in some places are alongside the normal trabeculae.

DISCUSSION

Paget's disease, or osteitis deformans, is not a common condition in India, although a few cases have been encountered. The spine often is involved as well as the other bones. The commoner sites of affection of the spine are the lumbar and the thoracic regions, the cervical region being less frequently involved.

Neurological complications of a milder type have been recorded in a number of cases. But paraplegia is not common. We found a total of 48 published cases of cord compression caused by osteitis deformans. Wyllie,[8] Kay *et al.*,[2] Schwarz and Reback,[4] Turner,[6] Whalley,[7] Teng *et al.*,[5] and Robinson[3] have reported cases. The commonest site for compression paraplegia in Paget's disease is the thoracic region. The interpeduncular spaces are the narrowest in the thoracic region between the 3rd and 10th dorsal vertebrae, and hence the cord is most likely to be compressed in the upper thoracic region. Paget's disease of the cervical spine with cord compression is very uncommon. Only 3 cases were found in the literature. Garcin *et al.*[1] presented a case of Paget's disease with involvement of C4 vertebra. The patient had sudden development of quadriplegia and later partly recovered. Schwarz and Reback[4] reported cervical cord compression at C6–C7 level in a patient with advanced Paget's disease. Whalley's[7] case has already been referred to.

The patient is usually a male in the fifth or sixth decade of life and the onset of symptoms is gradual and insidious. A noteworthy feature is that often it is the neurological picture that presents itself first and brings the patient to the doctor. The neurological complications are less often met with in the obvious cases of Paget's disease. Because the neurological disability presents itself primarily, there is often some difficulty in diagnosis. Especially in older people complaints like aches and pains, difficulty in passing urine, etc., may be attributed to other factors and careful examination of the functions of the spinal cord may not be carried out. This important point has been emphasized by Robinson.[3]

The spinal cord and nerve root symptoms usually occur when the overgrowth of bone is such as to reduce the capacity of the spinal canal or the intervertebral foramen. The symptoms start gradually and progress towards signs of full compression of the cord. Sometimes the symptoms may be caused by interference with the blood supply of the cord, resulting in ischaemia.[7] Whether osteitis deformans will diminish the local blood supply of the cord is a difficult point, as usually the affected bone is very vascular. The association of arteriosclerosis with Paget's disease may explain those cases in which there are bizarre neurological findings. The interference with vascular supply may also be caused by foraminal narrowing. The problem of interference with the blood supply of the cord in diseases of the vertebrae is being studied separately in relation to fluorosis and spondylitis.

Collapse of the vertebra from softening, with resultant kyphosis and cord compression, is another cause of paraplegia. Dislocation may occur leading to vertebral displacement. In the case reported by Whalley[7] in 1946 the spinal canal was narrowed because of the enlargement of the axis.

Platybasia or basilar impression is associated with Paget's disease of the atlas and axis. In these cases, the odontoid process, being at a higher level than normal, causes neurological signs, especially affections of the lower cranial nerves, nuchal stiffness, torticollis, etc.

In the case reported here the spinal canal was compressed between the enlarged odontoid process and the body and laminae of the axis. The operation was bloody because of extreme vascularity. This condition has been noted by others as well and makes surgery a difficult procedure.

Another interesting feature is the localization of the affection. Roentgenograms of other bones did not reveal any evidence of Paget's disease.

SUMMARY

A case of spinal cord compression caused by osteitis deformans of the axis is reported. No other bone of the skeleton was involved.

We wish to express our thanks to the Dean, General Hospital, Madras, for permission to publish this case.

REFERENCES

1. Garcin, R., Varay, A., and Dimo, H. Effondrement vertébral aigu au cours d'une maladie osseuse de Paget. Quadriplégie transitoire. Syndrome de Brown-Séquard, résiduel (contribution à l'étude des complications médullaires de la maladie de Paget). *Rev. neurol.*, 1937, *67:* 761–769.
2. Kay, H. D., Simpson, S. L., Riddoch, G., and Vilvandré, G. E. Osteitis deformans. *Arch. intern. Med.*, 1934, *53:* 208–248.
3. Robinson, R. G. Paraplegia due to Paget's disease (osteitis deformans). *Brit. med. J.*, 1953, *2:* 542–544.

4. Schwarz, G. A., and Reback, S. Compression of the spinal cord in osteitis deformans (Paget's disease) of the vertebrae. *Amer. J. Roentgenol.*, 1939, *42:* 345–366.
5. Teng, P., Gross, S. W., and Newman, C. M. Compression of spinal cord by osteitis deformans (Paget's disease), giant-cell tumor and polyostotic fibrous dysplasia (Albright's syndrome) of vertebrae. A report of four cases. *J. Neurosurg.*, 1951, *8:* 482–493.
6. Turner, J. W. A. The spinal complications of Paget's disease (osteitis deformans). *Brain*, 1940, *63:* 321–349.
7. Whalley, N. Paget's disease of atlas and axis. *J. Neurol. Neurosurg. Psychiat.*, 1946, n.s. *9:* 84–86.
8. Wyllie, W. G. The occurrence in osteitis deformans of lesions of the central nervous system. With a report of four cases. *Brain*, 1923, *46:* 336–351.

A NEW METHOD FOR APPLYING POLYETHYLENE FILM TO THE SKULL IN THE TREATMENT OF CRANIOSYNOSTOSIS

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The use of polyethylene film to retard the regrowth of bone following craniectomy for craniosynostosis has been established as an important feature in operative technique.[1,2] The technical problem of fixing the film in position has been managed formerly by punching holes along the margins of the bony defect and sewing the plastic in place with silk sutures. This part of the procedure frequently accounts for a significant portion of the operative time. The duration of the operation is a matter of importance in the small infants who are usually the subjects for craniectomy. For this reason it has been considered advisable to develop a more rapid and convenient method for the application of polyethylene film to the margins of the skull. This consists of stapling the plastic over the edge of the bone with specially designed tantalum clips.

TECHNIQUE

A clip applicator* has been made, which has a powerful action of the jaws, that works at a right angle to the shaft of the instrument (Fig. 1). The head of the applicator is small enough to be introduced easily into a craniectomy defect of 8 mm. The lower jaw has a thickness of 2.5 mm., thereby preventing undue stripping of the dura mater from the skull during the application of the clip. The jaws open to a maximum width of 7 mm. thus permitting its use even in older infants. The open width of the jaws can be regulated by means of a thumbscrew in the handle, allowing the clip to be held tightly in place while passing the instrument (Fig. 2).

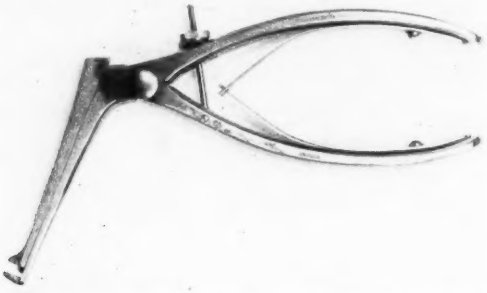

FIG. 1. Clip applicator. The knurled screw on the handle can be adjusted to fix the open width of the jaws.

* Available through Codman & Shurtleff, 104 Brookline Ave., Boston, Mass.

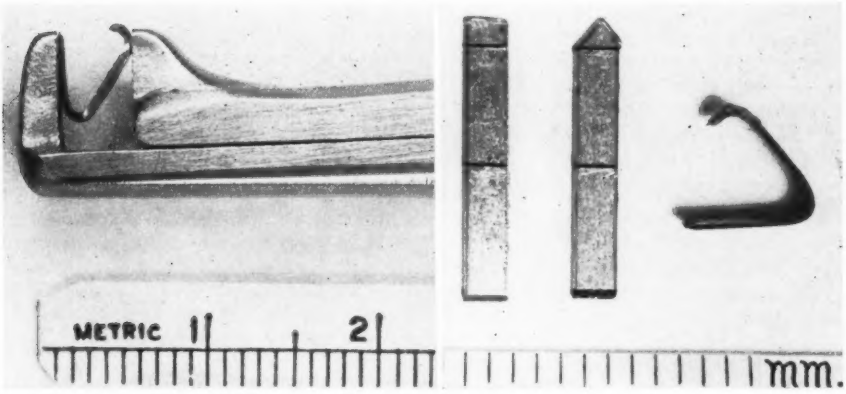

FIG. 2 (*left*). The clip may be held in the applicator while the instrument is being passed. The lower side of the clip is placed flat against the jaw of the applicator for greater stability.

FIG. 3 (*right*). The clips are cut out of 2 mm. strips of 0.015″ tantalum. This size is easily cut with small wire cutters.

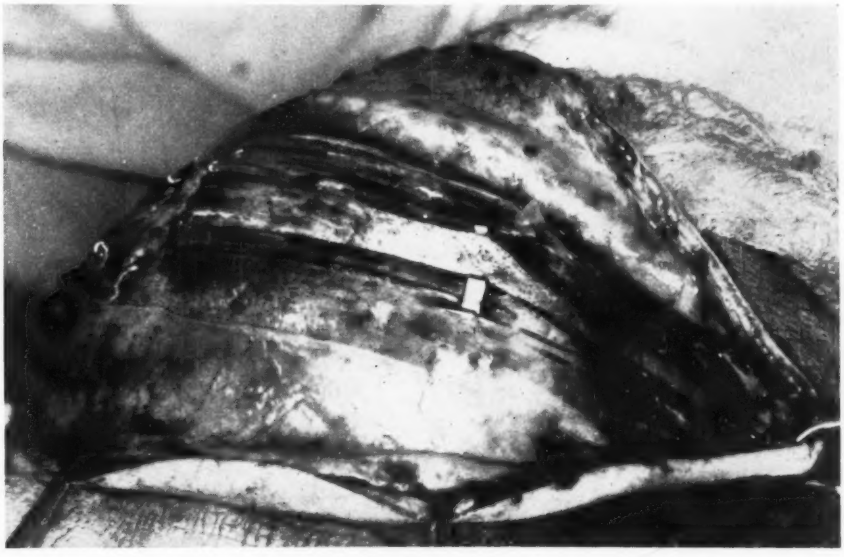

FIG. 4. Coronal craniectomy showing the polyethylene film applied to the edges of the bone by means of the tantalum clips.

The clips are made from tantalum which is 0.015″ thick. They can be fashioned by having a sheet of the metal cut into strips 2 mm. in width. These strips can then be cut by the nurse prior to operation to makes clips of the desired size. We have found that the most satisfactory design is to have the clip pointed at only one end. The pointed end is used to penetrate the outer table of the skull and hold the clip securely in place. The straight side lies flush with the inner table of the skull and does not depress the dura mater.

The clip is made from a 12-mm. length of the tantalum strip with the point beveled 1 mm. from the end. Regular wire cutters can be used to cut the metal. The point is then bent at a right angle to the strip and a second bend is made in the middle of the clip (Fig. 3). This may be modified depending on the thickness of the bone. It has been found that such a clip is about the right size for children under 1 year of age (skull less than 4 mm. thick). It will hold very well in the infant skull and the strength of the tantalum is such that the shape of the applied clip is maintained.

By using this method we have found it to be easier and faster to apply polyethylene film in these patients. The film is held smoothly and firmly in place and the operative time is significantly diminished (Fig. 4). Up to the present time 33 patients have been treated by this technique. There have been no complications incident to the use of this method and the development of the head following operation has been satisfactory in all cases.

REFERENCES

1. Ingraham, F. D., Matson, D. D., and Alexander, E., Jr. Experimental observations in the treatment of craniosynostosis. *Surgery*, 1948, *23:* 252–268.
2. McLaurin, R. L., and Matson, D. D. Importance of early surgical treatment of craniosynostosis. Review of 36 cases treated during the first six months of life. *Pediatrics*, 1952, *10:* 637–652.

BOOK NOTICE

Follow-up Studies of World War II Injuries. The U. S. Government Printing Office is publishing four volumes of some interest to Neurosurgeons. These include the National Research Council-Veterans Administration monograph on Peripheral Nerve Regeneration, the National Research Council-Veterans Administration monograph on The After-Effects of Head Injuries Sustained in World War II, and the Army Neurosurgical History of World War II in two volumes. The distribution lists are rather limited, but the sales price is fixed at cost.

The study on nerve injuries has just been published: Peripheral Nerve Regeneration. A Follow-up Study of 3,656 World War II Injuries. B. Woodhall and G. W. Beebe, Ed. Washington, D. C.: U. S. Government Printing Office, 1957, xxiv, 671 pp. Price $3.75.

Proceedings of Second International Congress of Neuropathology. At the request of the Organizing Committee of the Second International Congress of Neuropathology held in London, 1955 all reports, communications and discussions presented to the Congress have been published by the Excerpta Medica Foundation under the title: Proceedings of the Second International Congress of Neuropathology, London, 1955. These Proceedings consist of three parts. Part I and Part II contain 650 pages of text; Part III contains 156 plates, giving all illustrations to the text. This publication has been edited by Dr. W. H. McMenemey. The Proceedings may be ordered from the Excerpta Medica Foundation, 111 Kalverstraat, Amsterdam, Holland, at the price of £4/10/–, Fl. 50, or $12.50.

NOTICES

AMERICAN COLLEGE OF SURGEONS

The 43rd Clinical Congress of the American College of Surgeons will be held in Atlantic City, New Jersey, October 14–18, 1957. Two Surgical Forum sessions on October 17 at 8:30 A.M. and 1:30 P.M. will be devoted to reports of research in neurosurgery and neurophysiology. On October 18 at 9:00 A.M. there will be a symposium on "Dermoid and Epidermoid Tumors of the Central Nervous System," Dr. Edgar F. Fincher presiding, and at 10:30 A.M. a panel discussion on "Traumatic Intracranial Hemorrhage," Dr. E. S. Gurdjian, moderator. At 2:00 P.M. clinical neurosurgical papers on various subjects will be presented, Dr. Russell Meyers, presiding.

AMERICAN NEUROLOGICAL ASSOCIATION

At the 82nd annual meeting of the American Neurological Association held in Atlantic City, New Jersey on June 17–19, 1957, the following officers were elected for the coming year: President, Israel S. Wechsler; President-Elect, Bernard J. Alpers; 1st Vice-President, Russell N. DeJong; 2nd Vice-President, Bronson S. Ray; Secretary-Treasurer, Charles Rupp; Assistant Secretary, William F. Caveness.

The 83rd annual meeting of the Association will be held at the Claridge Hotel, Atlantic City, June 16–18, 1958.

ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASE

The annual meeting of the Association for Research in Nervous and Mental Disease will be held on December 13 and 14, 1957 at the Hotel Roosevelt, New York City. The subject of the meeting will be "The Effect of Pharmacologic Agents on the Nervous System."

BRAZILIAN NEUROSURGICAL SOCIETY

The first meeting of the newly formed Brazilian Neurosurgical Society will be held in São Paulo, Brazil on December 14, 1957. The President is Dr. José Ribe of Portugal, and the Secretary is Dr. José Geraldo Albernaz.

HOUSTON NEUROLOGICAL SOCIETY

The Houston Neurological Society, Houston, Texas, announces a symposium on "Pathogenesis and Treatment of Parkinsonism" to be held on March 13, 1958 as part of its Sixth Annual Scientific Session. Participants will include: Dr. Malcolm Carpenter, New York City; Dr. Harry Kaplan, Brooklyn; Dr. Arthur Ward, Seattle; Dr. Robert Schwab, Boston; Dr. Russell Meyers, Iowa City; Dr. Paul Bucy, Chicago; Dr. Irving Cooper, New York City and Dr. A. Earl Walker, Baltimore.

INTERNATIONAL CONGRESS OF NEUROLOGICAL SURGERY

At the International Congress of Neurological Surgery held in Brussels, Belgium, from July 21 to 27, 1957, the Executive Committee composed of delegates from each

of the member societies met. The Constitution which had been circulated earlier was adopted with the following changes:

The name of the organization was changed to "*World Federation of Neurosurgical Societies*," and the section on representation was adopted tentatively with the understanding that this matter would be reconsidered at the next Congress but that any proposed changes from the present Constitution must be submitted to all of the member societies twelve months in advance of the next meeting.

The Executive Committee chose the United States as the place for the next International Congress of Neurological Surgery, with the understanding that the meeting would be held on the Atlantic seaboard in the month of October in 1961.

The following officers of the World Federation of Neurosurgical Societies were elected: President, Paul C. Bucy, Chicago; Vice-President, E. H. Botterell, Toronto; Secretary-General, Bronson S. Ray, New York; Assistant Secretary-General, William B. Scoville, Hartford, Conn.; Treasurer, Barnes Woodhall, Durham, N. C.; Assistant Treasurer, Wallace B. Hamby, Buffalo, N. Y.; and Editor of Transactions, David L. Reeves, Santa Barbara, Calif.

These officers constitute the Administrative Council of the Federation and are charged with the responsibility of arranging for the International Congress of Neurological Surgery to be held in 1961. The exact time and place for the Congress will be selected by the Administrative Council and announced in the near future.

WESTERN NEUROSURGICAL SOCIETY

The Western Neurosurgical Society, which was founded on February 25, 1955 at Pebble Beach, California, held its second regular meeting in Portland, Oregon, December 9–11, 1956. The following officers were elected: President-Elect, Frank Turnbull; Vice-President, Howard A. Brown; Secretary-Treasurer, H. G. Crockett. The third regular meeting will be held at the Holiday Hotel, Reno, Nevada, September 29 to October 1, 1957.

MEETINGS

American Academy of Neurological Surgery, The Cloister, Sea Island, Georgia, November 11–13, 1957. Dr. Eben Alexander, Jr., Bowman Gray School of Medicine, Winston-Salem, N. C., Secretary.

American College of Surgeons, Atlantic City, New Jersey, October 14–18, 1957. Neurosurgical sessions on October 17 and 18. Dr. E. S. Gurdjian, 801 David Whitney Bldg., Detroit 26, Mich.

American Neurological Association, Claridge Hotel, Atlantic City, New Jersey, June 16–18, 1958. Dr. Charles Rupp, 133 South 36th St., Philadelphia 4, Pa., Secretary.

Association for Research in Nervous and Mental Disease, Hotel Roosevelt, New York, N. Y., December 13–14, 1957. Dr. Rollo J. Masselink, 700 West 168th St., New York 32, N. Y., Secretary.

Brazilian Neurosurgical Society São Paulo, Brazil, December 14, 1957. Dr. José Geraldo Albernaz, Secretary.

Congress of Neurological Surgeons, Statler Hotel, Washington, D. C., November 7–9, 1957. Dr. Philip D. Gordy, Suite B-1, Professional Bldg., Wilmington, Del., Secretary.

Harvey Cushing Society, Mayflower Hotel, Washington, D. C., April 21–23, 1958. Dr. David L. Reeves, 316 West Junipero St., Santa Barbara, Calif., Secretary.

Neurosurgical Society of America, Key Biscayne, Florida, January 15–18, 1958. Dr. Frank P. Smith, 260 Crittenden Blvd., Rochester 20, N. Y., Secretary.

Pan-Pacific Surgical Association, Honolulu, Hawaii, November 14–22, 1957. Dr. F. J. Pinkerton, Room 230, Young Building, Honolulu, Hawaii, Director General.

Western Neurosurgical Society, Holiday Hotel, Reno, Nevada, September 29–October 1, 1957. Dr. H. G. Crockett, 511 South Bonnie Brae St., Los Angeles 57, Calif., Secretary.

JOURNAL OF NEUROSURGERY

The *Journal of Neurosurgery* is devoted to the publication of work relating primarily to neurosurgery, including studies in clinical neurophysiology, organic neurology, ophthalmology, radiology and pathology. Short articles on unusual cases and on technique, special instruments or equipment that might be useful to others in the field are also acceptable.

MANUSCRIPTS are accepted with the understanding that they are contributed solely for publication in this *Journal*. They should be addressed to Dr. Louise Eisenhardt, 333 Cedar Street, New Haven 11, Connecticut, U.S.A. They should be typewritten in double space, with tabular matter and passages for small print on separate sheets. All materials should be mailed flat.

REFERENCES should be arranged alphabetically and cited by superior numerals in the text. The following order should be used:

Articles: Name of author, full title of article, name of periodical, year, volume, first and last page of article. Abbreviations of names of periodicals should conform to the style used in *World list of scientific periodicals published in the years 1900–1950* (London: Butterworths Scientific Publications, 1952).

Books: Name of author, full title of book, city, publisher, year, edition, full pagination (if book is in more than one volume, give the number of volumes).

ILLUSTRATIONS. Care should be taken not to use too many illustrations. All figures approved by the Editorial Board are provided free of charge with the exception of color plates which are inserted at the expense of the contributor. Permission must be obtained from the subject for reproduction of identifiable photographs of patients. If illustrations are larger than 8.5×11 inches, reduced glossy prints should be submitted instead. Any lettering should be large enough to permit necessary reduction. Paper clips should not be used on prints. A hard pencil should not be used in writing on the back of prints. Illustrations should be numbered and the top plainly indicated. Illustrations should be submitted UNMOUNTED.

SPECIAL TYPOGRAPHY. Tables and also text set up in small print in excess of 10 per cent of composition costs are charged to the author.

PROOF CORRECTIONS will be allowed without charge up to 5 per cent of the original cost of composition. The cost of changes in page proof, if made by the author, will be borne by him. *Page proof* will be submitted to authors only when special questions arise.

REPRINTS. The *Journal* supplies 50 offprints free to the senior author of each article. Additional offprints and reprints may be purchased according to a schedule of prices that accompanies galley proof.

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